UNIVERSITY OF TORONTO MEDICAL JOURNAL

CLINICAL EMERGENCIES

VOLUME XXXVIII

NUMBER 3

JANUARY 1961

THE INTERNAL CLOCK

The ability to judge intervals of time is a basic mental function. In order to learn how drugs affect this "internal clock," SK&F scientists use a test in which a monkey must hit a lever after an interval of 20 seconds to obtain food.

When he is under the influence of certain drugs, "time flies" and he misses the pay-off period by hitting the lever too late. Other drugs make "time drag" and he misses the pay-off period by hitting the lever too soon.

Because one of the characteristics of many mental disorders is a distortion of the time sense, observing how drugs affect this primary psychological process may reveal valuable information that will help SK&F scientists in discovering effective treatments for mental illness.

SMITH KLINE & FRENCH • MONTREAL

pioneering in pharmaceuticals . . .
for better health

INDEX

EDITORIAL 106

CLINICAL EMERGENCIES

HEMORRHAGIC SHOCK, DIAGNOSIS & TREATMENT, *E.F. Saunders, 6T1.* 107

ACUTE ARTERIAL OCCLUSION OF THE EXTREMITY, *A. Gardner, 6T1.* 111

ACUTE CRANIOCEREBRAL INJURIES, *N.M. Wolfish, B.A., 6T1.* 113

MEDICAL SHOCK, *L.M. Jerry, 6T1.* 117

CARDIAC EMERGENCIES, *P.A. Korhonen, 6T2.* 121

ACUTE PULMONARY EDEMA, *B. Fogel, B.A., 6T1.* 128

STATUS ASTHMATICUS, *G.J. Donsley, 6T1.* 129

RESPIRATORY DISTRESS IN INFANTS, *J.D. Gossage, 6T1.* 132

THE ACUTELY DEHYDRATED INFANT, *P.I.V. Truscott, 6T2.* 136

ECLAMPTOGENIC TOXEMIA, *D.J. McDonald, 6T1.* 138

VAGINAL HEMORRHAGE IN LATE PREGNANCY, *L.M. Jerry, 6T1.* 139

THREE OBSTETRICAL EMERGENCIES, *C.R. Swayze, 6T1.* 145

SUICIDAL RISKS, *M.A. Evans, B.A. 6T2.* 146

BOOK REVIEW 150

THE COVER

Entitled "The Harlot's Progress", this painting is the last of a series on the subject by Hogarth. While the physicians argue, the patient dies and "the two quacks, whose injudicious treatment has probably accelerated her death, are vociferously supporting the infallibility of their respective medicines, each charging the other with having poisoned her". This quote is from Hogarth's annotator, The Reverend J. Trusler, who went on to say that "These disputes, it has been affirmed, sometimes happen at a consultation of regular physicians, and a patient has been so impolite as to die before they could determine the name of his disorder". The lady plundering the trunk of the victim's possessions is a nurse typical of the times.

L.E.S.

INDEX TO ADVERTISERS

SMITH, KLINE & FRENCH	INSIDE FRONT COVER
ONTARIO RETAIL PHARMACISTS' ASS'N	page 104
HOMEWOOD SANITARIUM	page 106
PARKE-DAVIS	page 126, 127
FROSST	page 135
OSBORNE'S PHARMACY	page 149
DOWNS	page 150
ASTRA	INSIDE BACK COVER
POULENC	OUTSIDE BACK COVER

The MD's right-hand man...

The skilled retail pharmacist and his dispensary are always at your service. Even during the late hours of the night or early hours of the morning, he will dispense your urgent prescriptions, should an emergency arise.

He keeps himself well informed on the latest pharmaceutical information, and maintains a complete stock of up-to-the-minute drugs. He is always ready to serve you . . . to be your right-hand man.

ONTARIO RETAIL PHARMACISTS ASSOCIATION

THE ONTARIO RETAIL PHARMACISTS' ASSOCIATION

UNIVERSITY OF TORONTO MEDICAL JOURNAL

ESTABLISHED DECEMBER 1923
AN UNDERGRADUATE PUBLICATION

NUMBER 3
VOLUME XXXVIII
JANUARY 1961

editor-in-chief

L. E. SOPER, 6T1.

editorial consultants

M. BARKIN, M.D. — P. B. DENT, M.D

associate editors

J. R. ALLEN, B.A., 6T1. — B. FOGEL, B.A., 6T1.
P. C. SAMU, 6T1. — J. J. MOGAN, M.A., 6T1.
C. G. CHAMBERLAIN, 6T1.

assistant editors

G. E. HOWEY, B.A., 6T2. — J. SOBRIAN, 6T2.
E. S. LILKER, 6T2. — W. O. R. SPITZER, 6T2.
D. P. SMITH, 6T2. — B, W. THOMAS, 6T2.

feature editors

abstracts and microabstracts.....J. H. GOLDIE, 6T1.
C. E. BAYLISS, 6T1.
book reviews..... R. BAUMAL, 6T3.
A. H. GERULATH, 6T3.
on inspection..... L. M. JERRY, 6T1.

managing editors

Miss R. S. SILVERSTEIN, 6T5. — K. H. SHUMAK, 6T5.

business managers

J. L. MADIGAN, 6T3. — T. D. C. MARSHALL, 6T3.

advertising department

W. S. CRYSDALE, 6T4. — R. N. FOWLER, 6T4.
Miss P. C. WHILLANS, 6T4. — Miss K, J. WYTOCK, 6T4.
EWE GHEE GOW, 6T4.

circulation

J. D. GRAHAM, 6T3, *manager*

R. BALL, 6T3. — F. LANGER, 6T4.

art and photography

Mrs. M.E. SOPER — R. S. GILDER, B.A.

This journal is published six times a year from November to April, by the Medical Society of the University of Toronto, to serve primarily as a medium for student expression.

Subscription rates: $4.50 for one volume of six consecutive issues. Single copies $.75 plus $.10 postage.

Manuscripts including references and footnotes should be typewritten double spaced on one side of the paper. References should be kept to a minimum, and should be cited numerically in order in which they appear in the text.

Illustrations are included by arrangement with the Editor. Drawings should be done on white paper in black ink.

Final Copy Date is the 15th of the month prior to issue. Mailing date is the 15th of the month of issue.

Material printed in this Journal is covered by copyright. Papers are accepted for consideration with the understanding that they are contributed for publication in this Journal only.

Correspondence should be addressed to Room 114,
Medical Building,
University of Toronto,
Toronto 5, Ontario.

Litho by Chromo Litho.

Authorized as Second Class Mail,
Post Office Department, Ottawa.

EDITORIAL

One of the innovations the editorial staff has introduced this year is the devotion of the greater part of each issue to a specific topic of interest for medical students. This has been done in an effort to make our Journal more useful to the students as a source of readily found, "lean" material for the preparation of seminars, therapeutics problems and the other medical hurdles which we face.

This issue concentrates on the most pragmatic aspect of medical practice: how to deal with emergencies. The benefit derived from digesting, absorbing and retaining the information in this issue is obvious; the Journal will not be accessible when the emergencies arise.

We define emergency as a clinical situation that demands the immediate attention of a physician in order to avoid irreversible damage in the patient. It is our hope that when you find yourself deeply involved in the highly recurrent emergency of facing a stern, implacable clinician across the bed in an oral, some of the facts you find in this issue may save you from irreversible damage.

W.O.R.S.

Homewood Sanitarium

A private hospital for the diagnosis, care and treatment of all types of psychiatric disorders. An adequate staff of specialists study each patient carefully and fit the treatment to his individual needs. Excellent cuisine, comfortable accommodation and personal attention of specially trained nursing staff contribute to the recovery of the patient. All types of modern therapy are available, including Psychotherapy, Insulin and Electro-shock, combined with excellent Occupational and Recreational facilities. The Sanitarium buildings, accommodating 225 patients, are situated in a very attractive setting within the confines of the City of Guelph.

Homewood has been approved as a private hospital under the Ontario Hospital Insurance Plan. Benefits depend on type of illness and other factors. For full information write or telephone (TAylor 4-1010).

A. L. MacKINNON, M.B., Medical Superintendent

THE HOMEWOOD SANITARIUM OF GUELPH, ONTARIO, LIMITED

HEMORRHAGIC SHOCK, DIAGNOSIS & TREATMENT

E.F. SAUNDERS, 6T1.

Shock is a syndrome caused by sympathetic overactivity in compensation for "peripheral circulatory failure (which results) from a discrepancy in the size of the vascular bed and the volume of the intravascular fluid". — *Minot and Blalock;* Ann. Surg. 112, 1940.

CLASSIFICATION

1. Neurogenic
2. Hematogenic
3. Vasogenic
4. Cardiogenic

HEMATOGENIC SHOCK

Loss of Whole Blood — obstetrical and gynecological, gastro-intestinal, wounds, fractures.

Loss of Plasma — burns, peritonitis.

Loss of Electrolytes — burns, pyloric obstruction, intestinal obstruction, intestinal fistulae, diarrhea, diabetic acidosis, Addison's disease, low sodium syndrome.

ETIOLOGY

The primary cause of the shock syndrome is a loss of blood from the circulation. Hemorrhage may occur into tissues and body cavities, or to the exterior. The amount of blood loss necessary to produce shock varies with the individual, his age and previous physical status. A loss of one third of the blood volume will produce slight shock; a loss of two thirds, severe shock.

Predisposing Factors

Chronic shock: Many old people are in a state of "chronic shock". This means that they have a chronic hypovolemia due to hypofunction of the adrenal cortices or to chronic debilitating disease.

Anemia: Patients with a low hemoglobin level due to neoplasm, pernicious anemia or chronic disease are more prone to develop shock than a person with a normal hemoglobin. The effects of shock are more severe as the anemic patient starts with a decreased oxygen carrying capacity.

Peripheral Vascular Disease: The vessels of patients with marked arterial and arteriolar sclerosis can not react as efficiently as normal in compensating for blood loss.

Age: Due to the above three factors the geriatric patient is at grave disadvantage in responding to blood loss and suffers from a relatively higher morbidity and mortality.

Anesthesia: Most general anesthetics result in a lowered blood pressure. The use of muscle relaxants is an additional factor in producing hypotension. The combination of fluothane and curare is particularly known for its hypotensive qualities. In the patient who has lost blood, anesthesia may initiate the shock syndrome.

Perpetuating Factors

Certain factors are thought to perpetuate shock. They are: toxins from tissue breakdown, toxins from gastro-intestinal bacteria, endocrine factors, nervous factors.

PHYSIOLOGY

The classic manifestations of the shock syndrome result directly from the abnormal physiology of the circulatory system as it attempts to compensate for a reduced blood volume. Because of the hypovolemia the total oxygen carrying capacity of the circulation is reduced. The organs most likely to be damaged by anoxia are the heart and brain. The signs and symptoms are those of overactivity of the sympathetic nervous system.

Mechanisms

Vascular: The reduced blood volume of shock produces a decreased venous return, resulting in a decreased cardiac output, tending to produce hypotension. There is stimulation of the baroreceptors of the carotid sinus and aortic arch which reflexly increase respiratory and heart rates, and cause redistribution of the circulation. Arterioles constrict to reduce blood flow to skin, kidney, liver and splanchnic area. Large veins constrict to reduce total capacity of the circulatory system. There may be active dilation of vessels in the brain and heart. The above is "compensated" shock, exhibiting tachypnea, tachycardia, cold pale skin and normal or even increased blood pressure. If the carotid sinus and aortic arch reflexes are not present, a 10% blood loss can cause death.

Hormonal: Any injury to the body sets up the "stress reaction". This reaction is due to an increased production of adrenal and pituitary hormones. Adrenalin produces the most obvious effects since it reinforces the above changes in circulation and respiration. Recently a factor produced by the anoxic kidney has been found. It is called V.E.M. — vaso-excitor material — and it produces generalized vasoconstriction.

Reversible vs. Irreversible Shock: Shock is also divided into reversible and irreversible stages. When treatment is delayed, the compensatory mechanisms fail and even with transfusion of several times the original blood loss, death results. The vasodilatation found in irreversible shock results from anoxia of arterioles and capillaries. With relaxation of vasomotor tone there is a great increase in the capacity of the circulatory system and severe hypotension follows. Some workers think the liver produces a vasodilator material V.D.M. under anoxic

conditions and V.D.M. may play a part in the sudden vasodilatation. Time is a crucial factor in the irreversibility of shock. Experimentally, if an animal is bled and the blood replaced immediately, the animal lives. If there is delay in treatment, the replacement of blood must be greater than the original loss to maintain life. If there is a lapse of several hours, the animal dies regardless of the amount of blood transfused. Therefore, shock must be treated in the reversible stage. The time margin before shock become irreversible is decreased when complications are present.

DIAGNOSIS

It is most important to recognize the possibility of blood loss and shock. A list of specific conditions in which shock may develop follows below. The signs and symptoms of the shock syndrome vary with the acuteness and severity of blood loss. They will vary also with the time lapse following injury.

Early Signs and Symptoms: history of trauma, thirst, cold extremities, evidence of a wound and hemorrhage, deep, rapid respiration, restlessness, pallor, cold sweating skin, dry mouth, normal or increased blood pressure, decreased pulse pressure, normal or increased pulse rate.

Late Signs and Symptoms: Additional to above; delirium or unconsciousness, decreased or unobtainable blood pressure, rapid weak pulse — or absent pulse, collapse of veins, oliguria. The pulse is an extremely unreliable signpost in early shock. The classic thready rapid pulse is a late sign. Workers have found the pulse rate to be normal, increased or decreased. Contrary to expectation, in sudden severe hemorrhage the pulse rate tends to decrease.

The *blood pressure* is also unreliable in shock. In compensated shock, the pressure is normal or increased and only late in events does it fall. A decrease in pulse pressure is said to be of more value. Due to vasoconstriction, the diastolic pressure is increased and simultaneously a reduced cardiac output reduces systolic pressure. One must be on the alert for the previous hypertensive. In this patient, an apparently normal blood pressure may indicate severe shock. There is a test that may be of value in diagnosis. The patient can be put in a sitting position momentarily. There is a drop in blood pressure in the shocked patient. Conversely, the patient's legs can be raised to produce a rise in pressure. "The old concept that acute hemorrhage can be readily diagnosed by a rapid pulse and a low blood pressure is erroneous". (Shenkin)

SPECIFIC CONDITIONS

External Hemorrhage

Wounds Due to Trauma: Diagnosis is easy and seldom missed.

Operative Blood Loss: The signs of shock may be masked in the anesthized patient. Therefore, careful attention must be paid to the amount of blood lost. Hemorrhage can be estimated from the amount of blood in the suction bottle. Hemorrhage at operation is almost always underestimated. It has been recommended that the sponges and towels be weighed and the blood in the suction bottle be accurately measured to calculate blood loss.

Internal or Concealed Hemorrhage

The diagnosis of concealed hemorrhage is occasionally missed with disastrous consequences to the patient. The signs and symptoms are those of shock as well as those of the underlying condition.

Fractures: A fractured femur commonly causes shock due to blood loss. As early as 1937 it was demonstrated that "concealed hemorrhage into an injured part was far greater than casual inspection would indicate and large enough to be of definite significance as a causative factor in shock". An increase in the radius of the thigh of only 1 cm. indicates a loss of 2 to 2.5 litres of blood from the circulation. Immobilization of the fracture site as a first aid measure keeps hemorrhage to a minimum.

Intra- and Retroperitoneal Hemorrhage: Penetrating wounds, a blow, or a crush injury of the trunk can produce severe damage to abdominal organs with subsequent hemorrhage and shock. Abdominal contents may be torn or avulsed by the deceleration injury of an automobile accident. There may be no external signs of violence. Massive intra-abdominal hemorrhage produces characteristic signs of abdominal distension, rigidity, deep tenderness, shifting dullness, absent bowel sounds and nausea and vomiting as well as signs of shock.

Rupture of Spleen: Although there is usually a history of severe trauma to the upper left abdomen, mild trauma can rupture a previously diseased spleen (leukemia, malaria, infectious mononucleosis, Banti's syndrome). Rupture of the spleen is of two types. The *immediate* type is diagnosed when there is dyspnea, pain increased on breathing, pain in left shoulder, hyperesthesia in left shoulder — Kehr's sign, signs of intra-abdominal hemorrhage, and signs of shock.

Delayed rupture is due to a rupture of the parenchyma producing a subcapsular hematoma. Diagnosis is based on finding a mass in left upper quadrant and hypochromic microcytic anemia. The patient is usually sent home only to have the hematoma rupture into the abdominal cavity several days later producing the picture of an immediate rupture. The treatment is splenectomy in both cases.

Rupture of Liver: Injuries to the liver are equal in incidence to those of the spleen. Both organs may be injured simultaneously. Because of the vascularity of the liver, there is profuse hemorrhage. A bile peritonitis may occur. Other signs are similar to those of a ruptured spleen. A central rupture of the liver can bleed via the biliary tract into the duodenum producing vomiting of blood and tarry stools.

Injury to Small Bowel and Colon: Injury usually occurs at fixed points, the duodenal — jejunal flexure, the junction of ileum and cecum, and the proximal and distal ends of the sigmoid colon. The bowel may be torn, or severed from its mesentery and vessels. This will produce signs of intra-abdominal hemorrhage, of shock, of bowel obstruction, and of peritonitis. X-ray of the abdomen may show free air in the peritoneal cavity. It is not recommended that the shocked patient be X-rayed in the upright position for air under the diaphragm.

Injury to Mesentery: Tears of the mesentery involve the arcade of vessels causing severe hemorrhage. Rarely, an intra-mesenteric hematoma results. The bowel which has lost its blood supply becomes gangrenous. Diagnosis is the same as that of injury to the bowel itself.

Rupture of Kidney: Injury to the kidney is of four types; contusion, hematoma — Gerota's fascia intact, retroperitoneal hematoma, and avulsion of the pedicle. Diagnosis is made where there is pain, swelling and ecchymosis in the flank with dullness over kidney, hematuria, abdominal mass and signs of shock. An intravenous pyelogram must be done. It may show extravasation of dye, loss of psoas shadow and lateral deviation of the spine. Nephrectomy is the treatment for severe hemorrhage provided there is a normal, functioning kidney on the other side.

Post-operative Hemorrhage: In association with abdominal prodedures, intraperitoneal hemorrhage can occur in the first 24 hours post-operatively and is due to slipping of a ligature on an artery. Fortunately, it is rare. Diagnosis is difficult. The signs are those of shock. When in doubt the abdomen must be reopened.

Spontaneous Gastro-intestinal Hemorrhage

Massive bleeding of the gastro-intestinal tract is usually due either to a peptic ulcer or to esophageal varices.

Hemorrhage is the most frequent cause of death from peptic ulcer. A posterior duodenal ulcer eroding into the gastro-duodenal artery causes massive hemorrhage. There is an ulcer history in most cases. The patient vomits large quantities of blood and passes tarry stools. Esophageal varices produce massive hematemesis. The patient is usually a known alcoholic and has other signs of portal hypertension.

Obstetrical Causes of Hemorrhage

Severe hemorrhage is a common complication of pregnancy and delivery and is still the primary cause of maternal mortality.

TREATMENT

1. *General*

Ensure an airway — a tracheotomy may be necessary — and stop the bleeding — direct pressure on an external wound is the best method of hemostasis. Internal hemorrhage may need operative intervention.

Survey of Injuries: This can be done quickly to note such entities as a tension pneumothorax, sucking wound of the chest or fracture of a major bone.

Venapuncture: If the veins are collapsed, a stab venapuncture is difficult and a "cut down" may be necessary. This is done on an ankle vein one inch superior and anterior to the medial malleolus. A polyethylene catheter can be used if long term intravenous therapy is anticipated. Take a blood sample for blood type, cross match and hemoglobin.

Administration of Fluids

Plasma Volume Expanders: Normal saline is a poor expander as it is quickly lost from the circulation and has no colloid osmotic pressure.

Three artificial plasma volume expanders are commonly used; dextran, polyvinyl pyrrolidine and oxypolygelatin. Dextran, a polymer of glucose is administered as a 6% solution in saline. One or two units can be given safely but more than one litre may cause a bleeding tendency. Dextran is a good expander remaining in the circulation longer than administered plasma. The length of action can be varied by regulating the molecular size. PVP is a polymer of formaldehyde and acetic acid. It is used in a solution isosmolar with plasma. PVP is not used much now as it appears to be retained by the body to some degree. It will also cause a bleeding tendency. Gelatin will not cause a bleeding tendency and lasts about as long as plasma. It is completely excreted by the kidney.

Salt-free, normal human serum albumin is used as a 5% solution in normal saline. There is no risk of serum hepatitis or citrate intoxication. A drawback is the expense of the preparation.

Plasma from single donors carries no greater risk of hepatitis that whole blood. The incidence of hepatitis virus carriers in blood donors is about 1 in 250. A crossmatch is needed for isoagglutinin titre. Plasma of known low titre can be used immediately without a crossmatch.

Pooled human plasma may be used. If 20 or more units of plasma are prepared together, a

crossmatch is unnecessary as the isoagglutinins are diluted. There is danger of serum hepatitis developing in all users of an infected pool. It has been found that storing pooled plasma for 6 months, at 30° to 32°C. will kill the hepatitis virus.

There is a danger of *citrate intoxication* with both plasma and whole blood. If administration is slow there is little danger but, with rapid transfusions, intravenous calcium gluconate should be given. It must be given separate from the infusion as the calcium causes clotting. As a routine, 1 gm. of 10% solution of calcium gluconate can be given for every 3 units of blood or 1.5 units of plasma infused in less that an hour. Plasma contains twice as much citrate as blood.

Whole Blood: Type-O Rh-negative blood is blood from a "universal donor". It should be tested for isoagglutinin titre and only blood of low titre used without a cross-match.

Blood of the patient's own type for transfusion must be checked by typing and a cross-match against the patient's blood. This process takes some time and it is during this interval that plasma volume expanders and universal donor blood are used. *There is no substitute for whole blood.* One unit only, should never be given because of the possible complications of transfusion. If one unit of blood suffices the patient, the blood was not needed in the first place. It has been found that 40% of transfusion recipients received only one bottle.

Two units of blood should be given in ghe first 10 minutes. If necessary, several veins can be used simultaneously and, in extreme cases, blood can be pumped into the patient under positive pressure. There is danger of air embolism with this last procedure. At least 2,000 cc. of blood should be given to a patient in moderate shock. As a unit contains only 400 cc. of blood, (the rest is citrate solution) at least 5 bottles are needed. If bleeding continues or hypotension is refractory, 5 to 10 litres of blood may be needed. The patient should be checked after each bottle. Transfusion is stopped when the signs and symptoms of shock are gone, the hemoglobin level is up and a urine output of at least 40 cc. per hour is maintained. The blood pressure and pulse return to normal before the blood volume does and one further bottle of blood should be given. It is unusual to give too much blood. If this occurs the patient develops dyspnea, increased jugular venous pressure and basal rales.

No potassium should be given because there is already an increased potassium level due to tissue breakdown. Added potassium, in combination with reduced renal function, may cause heart failure.

Drugs

Antibiotics have been given to control the bacterial factor in shock but their value is questionable.

ACTH and cortisone are of little value as the adrenal cortex is working at capacity.

Vasoconstrictors are not needed as vasomotor tone is already maximal.

Analgesia is not necessary in the apathetic or delirious state as no pain is felt. Morphine can be given to the alert conscious patient who is in pain. Morphine must never be given subcutaneously, always intravenously in the shocked patient. Because of decreased circulation to the skin, subcutaneous morphine is not absorbed and multiple doses can cause morphine poisoning when circulation to the skin is restored. *Sedatives* are of no value.

Supportive Measures

Heat: It has been taught in the past that the shocked patient should be kept warm. This treatment was based on the cold skin, although rectal temperatures are normal in shock. It seems that a body temperature of a degree or so below normal is beneficial. Addition of heat or several blankets to the patient causes vasodilatation in the skin and therefore diverts blood from vital centers.

Oxygen: may be of value.

Position: The classic position for the shock patient is one where the feet are raised with the foot of the bed on shock pins. This position is dangerous in obese patients or those with heart disease or peripheral vascular disease. It has been criticized as being of no value and it is stated that the recovery of normal postural vasomotor reflexes is delayed.

Charting: A fluid balance chart should be kept showing oral and intravenous intake, and urinary output. It is most difficult to have nurses do this accurately. Temperature, pulse, blood pressure and respirations should be charted every half hour until the patient is out of danger.

Some Recent Concepts in Therapy

Intra-arterial Transfusion: It is generally concluded that intra-arterial infusion has no advantage over the intravenous route for transfusion. It is the rate of transfusion that is most important. There is a possibility of gangrene of a limb due to arterial spasm.

Ganglionic Blocking Agents: The reasoning behind the use of these is that vasoconstriction is prevented, reducing capillary anoxia and preventing increased capillary permeability. This prevents a further loss of fluid from the circulation. The therapy is not of proven value.

Hypothermia: This is used to minimize the effects

of anoxia on the brain and heart. Chlorpromazine or light pentothal anesthesia prevents shivering. This too is not a proven method of therapy.

FAILURE OF THERAPY

Occasionally it is found that a patient fails to respond to 2,000 cc. or more of blood. Therapy failure must never be attributed to irreversible shock. It is necessary to reassess the patient. The following may cause a refractory shock state; further concealed hemorrhage, pneumothorax, cardiac tamponade, head injury, spinal cord injury, peritoneal contamination, myocardial infarct, and error in diagnosis of the cause of shock.

SUMMARY

The presentation of a patient with a shock syndrome due to blood loss is a common occurrence. Diagnosis depends on recognition of the possibility of shock and on the general condition of the patient. The blood pressure and pulse rate are unreliable. Treatment is the rapid administration of whole blood in large amounts.

ACUTE ARTERIAL OCCLUSION OF THE EXTREMITY

A. GARDNER, 6T1.

The occurrence of an acute arterial occlusion is extremely dramatic. There is only a short period of time in which the attending phisician can effectively treat his patient and this is usually given as six hours. In the leg, once the affected muscles have become firm, tender and paretic and the ankle dorsiflexed, and once there is frank gangrene, amputation is imminent. We shall, therefore, be mainly concerned with the diagnosis and emergency treatment of these cases. As occlusion of the arteries of the lower limb occurs in higher incidence than that of the upper limb, we shall use the leg as our example.

CLINICAL PICTURE

The patient, more often than not, will complain of a sudden severe pain in the leg, followed quickly by numbness, coldness, and loss of function.

Examination will reveal an anxious, worried patient in acute distress. The leg will appear pale and the superficial veins will be collapsed. The limb will be cold to palpation, and a transition between this affected tissue and the proximal warm viable tissue will be noted. Distal to the point of occlusion the pulses will be absent, sensation will be diminished or absent, and active muscle movements will be minimal or nil.

These are the more common findings, and, of course, they will vary. In the occasional patient, pain may not be a factor and unless the physician is knowledgeable there may be an ill-fated delay in diagnosis. If the occluded artery is of large calibre the signs and symptoms of shock may be present, and this may distract the unwary.

DIFFERENTIAL DIAGNOSIS

The diagnosis of this condition is extremely important for the preservation of the limb. Occlusion may be either thrombotic, embolic, or vasospastic.

The latter may occur reflexly in acute thrombophlebitis. This condition generally follows surgical or obstetrical procedures, and such a history may be obtained. Usually there is a fever (101° to 103° F). The leg appears cyanosed and swollen, and the superficial veins are congested. If the calf veins are affected, Homan's sign (pain in the calf on forced dorsiflexion of the foot with the knee extended) is present. The differentiation between embolism and thrombosis is essential for in some cases the former necessitates surgery while the later precludes it.

"If the condition is due to an embolus, there is sudden occlusion in a patient who has no evidence of peripheral vascular disease but has some cardiac disease which furnishes an origin of the embolus". Probably the commonest cause is atrial fibrillation, and this is frequently associated with mitral stenosis. Embolic phenomenon are more prone to occur with conversion of the fibrillation to normal rhythm. Less commonly, emboli may originate from a mural thrombus on the wall of an infarcted left ventricle; or from a thrombus proximal to the site of obstruction on the wall of another artery or within an aneurysm. Also, paradoxical embolism, although rare, should be kept in mind. Embolism must always be considered if there is more than one acute peripheral occlusion.

If the condition is due to a thrombus there is sudden occlusion in a patient who shows no evidence of an embolic focus but who has some peripheral vascular disease, viz. intermittent claudication. As the commonest cause is arteriosclerosis obliterans, one would expect that the onset would be slightly more gradual.

The signs of thrombosis are not usually as

clear for any given level of obstruction as those of embolism. Embolic occlusion occurs characteristically at the bifurcation of the artery, because the distal branches are smaller than the main trunk. The level can be fairly confidently assessed by noting, on physical examination, the distal level of normally functioning tissue. For example, occlusion at the aortic bifurcation will affect both legs entirely; that of the common iliac, of one leg. If, on the other hand, the block occurs at the femoral bifurcation, the area below the knee will be affected with pain, coldness, and loss of sensation. Gastrocnemius and soleus will be involved, but quadriceps will be intact. With occlusion at the popliteal bifurcation, the foot will be affected. Since no muscles arise here, foot movements will still be possible. Generally, anastamoses around the site of occlusion are sufficient to supply that area. Thus the level of clinical findings occurs at the next distal joint, as outlined in the above examples.

Thrombosis, however, may occur anywhere along the length of the artery, and as a result, one would not expect, and one actually does not find, physical findings which are as definite.

It should also be remembered when differentiating embolism and arteriosclerosis obliterans, that the latter is more common in men than in women, more common in the elderly than the young, and more common in diabetics, in which case sex and age are not as important.

In some cases, the cause of occlusion cannot be determined on clinical grounds and arteriography is necessary.

TREATMENT

When a patient gives the above history he must be seen immediately and treated vigorously, as one's ability to salvage the leg declines rapidly after six hours of ischemia. At the home, the patient should be adequately sedated with intravenous morphine. In order to prevent thrombotic extension of the obstruction, anticoagulants should be started with 2 cc. of an intravenous heparin solution of concentration 1000 U/cc. One must attempt to reverse any reflex vasospasm in those collateral vessels not affected by the obstruction, and thus re-open essential alternate arterial routes. Hot water bottles applied to the trunk — *not* the affected leg — will give a good result. Once at the hospital, Priscoline gr. 1/2 I.V. is administered and a procaine block of the appropriate sympathetic chain performed.

The patient should be reassessed in about one hour and evaluated as a candidate for surgery. Of importance in this respect are the general condition of the patient, the effect of emergency treatment with regard to the sufficiency of collateral circulation, and the cause of obstruction (embolic or thrombotic).

A prerequisite for any form of therapy is, of course, an evaluation of the general condition of the patient. As atrial fibrillation is so commonly a cause of embolism, and coronary disease is so commonly associated with arteriosclerosis, and cardiac status in particular should be determined and if necessary (eg. cardiac failure) given preference over surgery. It is better to lose the limb than the life.

Embolic occlusion should be re-assessed after a trial of the above treatment. If the leg is improving, embolectomy is not urgent and conservative therapy is continued. Some arteries, if blocked, will not require surgery because adequate anastamoses with unoccluded vessels are sufficient to maintain good blood flow. In the leg, the anterior tibial and posterior tibial arteries, if occluded, usually do not result in severe ischemia because of plantar anastamoses. Thus treatment of obstruction at these sites is expectant. If the leg is not improving on non-operative treatment, then embolectomy is necessary.

It must never be forgotten, that the embolus had an origin. Therefore one is not finished until further emboli are prevented by appropriate measures (eg. mitral commissurotomy).

The decision to operate on an acute thrombotic obstruction will be determined by the extent of arteriosclerosis. This is demonstrated by arteriography. If the involved vessels show widespread involvment or the outflow distal to the segment is poor, one can hardly expect a good result. Some have attempted thromboendarterectomy, but results have not been encouraging. Thus, with arteriosclerosis obliterans, unless expectant therapy is successful, amputation is forthcoming.

Upper Limb

As regards occlusion in the upper limb, the treatment is vigorously conservative. Because of anastamoses at the scapula, elbow, and palm, the vascular load is adequately supported when brachial, radial, or ulnar occlusion occurs. Thus embolectomy in the arm is unnecessary. Arteriosclerosis in the upper arm is very rare.

SUMMARY

A patient complaining of sudden pain in a limb which is found to be pulseless, pale, paretic, paraesthetic or numb, and cold, with collapse of superficial veins has an acute arterial occlusion of that limb and must be treated immediately and vigorously. Sedation, (morphine), anticoagulation (heparin), and vasodilatation (heat to the trunk — *not* the leg — Priscoline, and procaine sympathetic block) are begun at once. Emboli must be assessed as regards the efficiency

of collateral circulation. Arteriosclerosis obliterans often precludes surgery.

ACKNOWLEDGMENT — I would like to thank Dr. D. Wilson for his enthusiastic help in preparing this paper.

ACUTE CRANIOCEREBRAL INJURIES

N.M. WOLFISH, B.A., 6T1.

Injuries to the head may be: acceleration injuries, penetrating injuries or crush injuries. They may be trivial requiring minimal therapy and consequent discharge (with the understanding that any change in the *status quo* should be immediately reported), or may be severe and require hospitalization.

The pathological conditions that follow head injury may be classified as:

Scalp a) contusion
b) lacerations
c) hematoma

Skull fracture — open — closed
1. linear
2. comminuted
3. depressed
4. expressed
5. penetrating

Intracranial Contents
a) Brain
1. concussion
2. contusion
3. laceration
4. edema & increased intracranial pressure
5. subdural accumulation of C.S.F.
6. hemorrhage
- epidural
- subdural - acute
- subacute
- chronic
- subarachnoid - local
- generalized
- intraparenchymous - petechial
- massive
7. acute subdural hygroma

b) Blood or C.S.F. discharge or brain discharge
1. ear
2. nose
3. nasopharynx

Infection
1. Osteomyelitis
2. Meningitis
3. cerebritis and cortical ulceration
4. brain abscess - epidural
- subdural
- intraparenchymous

Complications
1. pneumocephalus
2. spurious meningocele

Associated Injuries
1. fractures
2. spine injury
3. chest
4. abdominal

EXAMINATION

A clinical classification in the preliminary assessment of the patient is based on the level of consciousness, for this forms the basis of further management. Patients may be: Conscious when seen; Confused, semi-comatose or comatose; Severely comatose with decerebrate rigidity.

The Confused, Semi-Comatose or Comatose Patient

Assessment of the Level of Consciousness: This must be quickly assessed in order to establish a base line for future reference.

The ability of the patient to converse, his reasoning powers, insight, memory and understanding, behaviour, response to commands or stimuli, rationality, presence of absurd speech or movements, incontinence of bowel or bladder can be rapidly observed.

Assessment of Vital Signs for Base Line: In rapid expanding intracranial lesions the pulse rate falls, and blood pressure rises. This is due to pressure effects on the brain stem, stimulating vagal and vagopressor centres. They will not occur if the centres are damaged either at the time of injury or in the late stages of hippocampal or tonsillar herniation.

The Head: The scalp is examined for signs of trauma. The ears, external canals, drums and nose should be examined for evidence of blood or C.S.F. leak, signs of fracture to the paranasal sinuses, mastoid air cells or middle ear. Fractures of the anterior fossa or sphenoid sinus may be suggested by nasal or nasopharyngeal bleeding, in the absence of facial injury.

Pupils: the size, shape, regularity, reaction to light of both pupils is compared. The development of a fixed dilated pupil, unresponsive to

light (either direct or consensual) indicates damage to the III Nerve on the same side. This may be due to stretching by a herniated hippocampal uncus, hemorrhage into the midbrain in the area of the oculomotor nuclei, avulsion of the nerve from the midbrain or injury to the nerve due to fracture of the basisphenoid. The larger normal pupil on the opposite side of a Horner's pupil should be differentiated from the fixed dilated pupil of III Nerve injuries. This is differentiated by pupillary reaction to light.

Ophthalmoscopic examination of the fundi is important in order to detect papilledema, evidence of increased intracranial pressure. However it is uncommon to detect papilledema before the first 24 — 48 hrs., even in the presence of a fatal increase of pressure.

III, IV, VI, Nerves may be assessed in the unconscious patient by sharply flicking the hand at the margins of the eye fields and noting whether the eyes move toward the stimulus. Inattention and deep coma must be differentiated from paralysis.

V Nerve corneal reflex is attempted in order to ascertain the functioning of the V and VII Nerve reflex arc. An absent reflex may be obtained in brain stem damage, fractures of the petrous temporal with injury to the V and VII Nerves.

VII Nerve paralysis may be readily diagnosed by simple observation, or by the degree of movement in facial emotions and expressions. In semi-comatose and comatose patients, reaction to pressure on the supraorbital nerve is a valuable guide. Facial paralysis is usually delayed in onset unless the nerve is involved at the time of injury by a petrous temporal fracture; otherwise, it is usually a result of cortical contusion.

VIII Nerve damage may be recognized by persistent unidirectional nystagmus, indicative of vestibular nerve damage.

Gag reflex should not be evoked but observed, for attempts to elicit this response may result in unwanted vomiting with consequent aspiration. A loss of swallowing and cough reflexes indicates a loss of brain stem functions.

Motor Survey

Power may be assessed by the degree or paucity of movement; by the falling away of the raised arm and the response to painful stimuli. Tone is tested in the usual manner. Reflexes are elicited and compared. Babinski responses may be obtained shortly after injury producing semi- or complete coma but can become flexor as general cerebral function improves. Nuchal rigidity. is tested to detect meingeal irritation but must be differentiated from cervical spine injury and generalized or decerebrate rigidity.

Extradual and subdural hematomata spread diffusely to involve an extensive area of brain surface and thus pressure is generalized rather than localized to a single region. Paralysis occurs only when pressure is sufficient to cause either hemispherical ischemia or uncal hermiation. Increased reflexes on the affected side may be apparent before any obvious weakness.

Sensory Survey: Sensation is best tested by the response to painful stimuli. Hypo-or anesthesia must be differentiated from weak or paralyzed limbs.

The Patient with Decerebrate Rigidity

Decerebrate rigidity, due to a disorder in the midbrain and upper pons, must be differentiated from decorticate rigidity where the lesion involves the motor parts of the cerebral cortex or internal capsule.

Decerebrate rigidity is characterized by extension of all four extremities, opisthotonus and tonic neck reflexes (extension of the right arm and leg with flexion of the left arm and leg upon turning the head to the right); whereas decorticate rigidity is recognized by arm flexion, leg extension, or diagonal postures (arm and opposite leg flexed, remaining limbs extended).

The Conscious Patient

The examination is essentially the same but the survey may be easier and more complete with the co-operation of the patient. Particular attention must be paid to the level of consciousness, lucidity, irrationality etc. Specific complaints helpful in diagnosis may be obtained, i.e. head pain, headache, nausea, dizziness, photophobia.

INVESTIGATION

In addition to the history, physical, neurological examination, urine and routine blood analysis, various diagnostic aids are available. These include x-ray studies of the skull, lumbar or cisternal puncture, air studies (ventriculography or encephalography) angiography and electroencephalography.

X-rays are used to determine the presence, position and type of fractures, pneumocranium, the presence of foreign bodies, and if the pineal is calcified (50%), the presence of shift indicative of a space occupying lesion.

Lumbar puncture is a valuable diagnostic aid but should by no means be looked upon as a routine procedure. It is definitely contra- indicated in suspected epi- or subdural hematoma because of the danger of tentorial or cerebellar herniation. In carrying out this procedure attention is paid to the opening and closing pressures, the nature of the fluid and cytological and chemical studies carried out. The presence of blood or xanthochromia indicates cerebral contusion and laceration, i.e. subarachnoid hemorrhage. The concentration of the blood is a general

indication of the severity of the damage. Raised pressure often points to a space occupying mass, although normal or low levels can result.

Air studies are used for localizing lesions or ruling out their presence, ventriculography preferable to encephalography in the acute case. Those procedures may be dangerous especially marked swelling of the brain, for additional pressure caused by the injected air may be injurious or fatal in the first 10 — 20 hours.

Angiography is of value in the patient presenting with a syndrome of increasing deterioration of consciousness associated with focal manifestations.

E.E.G. is of questionable value in the acute lesion but is of great benefit in the chronic conditions.

INITIAL MANAGEMENT

The conscious patient when first seen may have suffered momentary unconsciousness and post-traumatic amnesia due to concussion. Ideally these people should be observed for at least 24 hours in order to detect the delayed onset of deterioration of consciousness or other complications. However, because of refusal on the patient's part to be hospitalized, or if the necessity of hospitalization is not urgent, discharge of the patient should be accompanied with the following instructions. The patient should return to Emergency at once if there is:

1) Increasing drowsiness
2) Difficulty in rousing the patient (every 2 hrs.)
3) Vomiting
4) Slow pulse
5) Continuous headache
6) Neck stiffness
7) Blood or clear fluid dripping from the ears or nose
8) Weakness in any limb
9) Convulsions

Complications that may arise in this group are extradural hematoma, post-traumatic meningitis and chronic subdural hematoma.

Obviously all minor or associated injuries are treated once it is assured that no serious neurological abnormalities are present. Scalp and minor wounds are adequately prepared and sutured as necessary.

In the more acutely ill patient the initial procedures are dependant upon the level of consciousness and co-operation. As the patient so warrants the following measures are indicated.

Maintenance of Aedquate Pulmonary Function:
Correction of Shock:

First Aid Procedures: Splinting of fractures, correction of bleeding wounds, chest or abdominal injuries may take precedence over the head injury.

Hydration: Hydration of the patient is important expecially if a C.N.S. disturbance has led to electrolyte water or glucose imbalance. The hydration rapidly develops and is complicated by starvation and restlessness. The use of a nasogastric tube provides a route for fluids (2.0 to 2.5L containing 2,000 to 2,500 calories a day) and the maintenance of an adequate urine output. If vomiting becomes a problem intravenous hydration may be necessary.

Catheterization: Following injury urinary retention or incontinence may be encountered necessitating the use of intermittent or indwelling catheterization.

Hyperthermia: Disturbance of brain stem function will, in addition to other manifestations cause an increase in temperature. Such patients should be lightly covered. sponged frequently, covered with wet sheets or ideally placed in a cool environment.

General Measures: Frequent positioning of the patient prevents bed sores, phlebothrombosis and hypostatic pneumonia, etc. Other measures in the care of the skin, bowels, bladder, exercises to prevent contractures and frozen joints must be undertaken.

If patients are unduly restless, the use of side rails and wrapping the hands in thick cotton wool bandages to prevent self injury are part of the general supportive measures.

Miscellaneous: The use of sedatives should be minimal because of their interference in vital signs. Morphine should never be employed due to its depressant action of the respiratory centre. It is important to determine whether the patient has been receiving previous medications and to continue such medications is necessary, i.e. insulin, Digitalis, steroids, anti-convulsants, etc. The use of antibiotics and sulfonamides is indicated in the presence of concommitant infections. In patients with C.S.F. otorrhea, rhinorrhea, or bleeding from the ear or nasopharynx, these drugs should be employed routinely because of the suspicion of compound fractures and to prevent the development of post-traumatic meningitis.

MINOR HEAD INJURY

Two degrees of disturbed consciousness can occur. First is the patient who was never unconscious and usually has insignificant consequences (although the possibility of fracture, epi- or subdural hematoma exists). Symptoms that develop at once or in a few days: headache worsened by stooping or exertion, dizziness, loss of confidence, nervousness, insomnia, depression and fatigue. The mechanism of these symptoms is unknown in regard to a normal neurological investigation.

The second is the patient who has temporarily

lost consciousness and is said to have suffered a *concussion.* This is defined as "a transient loss of consciousness due to head injury, instantaneous in onset, manifests widespread paralytic symptoms, does not exhibit structural cerebral injury and is followed by amnesia for the actual moment of impact". Thus there is loss of consciousness, loss of respiratory and vasomotor activity, loss of corneal reflex and motor reflexes centered in the pons and medulla. Death at this time is due to respiratory arrest or cardiac arrhythmia. Once consciousness is regained only retro- and anterograde amnesia exists.

Complications

Delayed Traumatic Collapse: The patient who after mild cerebral trauma falls unconscious for a few minutes only to regain a state of normalcy again, is probably experiencing a vasomotor syncopal attack.

Immediate Traumatic Paraplegia: Where both legs become temporarily weak, numb, sometimes exhibit bilateral Babinski signs and sphincteric incontinence, injury to the motor area is the probable cause.

Immediate Hemi- or Monoplegia: These may be due to a localized and direct contusion and laceration of brain tissue, with or without bloody CSF, following mild concussion.

Delayed Hemi- or Monoplegia: This usually signifies epi- or subdural hemorrhage or hygroma, thrombosis, or intracerebral hemorrhage.

Acute Drowsiness, Confusion, Headache, or Coma: This is due, presumably, to local or generalized cerebral edema.

MAJOR HEAD INJURY: (Unconscious since injury).

The patients generally exhibit one or more of the following pathological changes: laceration, contusion, subarachnoid hemorrhage, extradural hemorrhage and skull fracture. A favourable prognosis is more likely when the coma is less deep, reflexes, blood pressure, pulse and respirations are normal and no obvious neurological signs are found.

A fatal outcome is probable when the patient is moribund from the onset, coma is profound, limbs are flaccid and are flexic, corneal and gag reflexes are absent, pupils small and nonreactive, or dilated, unequal and fixed. There may be surgical shock. Breathing is stertorous, then feeble and irregular.

In patients whose prognosis is less bleak, coma becomes semi-coma, blood pressure, pulse and temperature stabilize. Muscular tone and reflexes return. A sudden rise in temperature, cyanosis, respiratory difficulty may, however, result in death. In the next two to three weeks, there still remains the risk of pneumonia, meningitis, epi- or subdural hematoma.

Death during the first twelve hours is usually due to direct brain injury; death occurring later is usually the result of intracerebral or subarachnoid hemorrhage, herniation of the temporal lobe, localized or generalized edema, epi- or subdural hematoma, meningitis or pneumonia.

LUCID INTERVAL

This group of patients have experienced brief initial coma followed by recovery of consciousness, or no loss of consciousness and thus neither concussion or contusion was present. The following conditions must be considered.

Acute Epidural Hemorrhage

This usually follows temporal or parietal fracture with consequent tear in the middle meningeal vessels, or the dural sinuses. A lucid interval of several hours or days follows a momentary loss of consciousness, and this is followed by the development of: headache, increasing in severity, vomiting, drowsiness, confusion, seizures, hemiparesis, hypereflexia, and Babinski sign. As coma deepens hemiparesis and the Babinski sign may give way to flaccid or spastic limbs with bilateral Babinski signs. There may be aphasia. Respirations become deep and stertorous, then shallow and irregular, then finally cease. The pulse is slow and bounding and the systolic blood pressure rises. The pupil may dilate on the affected side. The C.S.F. usually is under increased pressure but may be normal or subnormal; the fluid may be clear or sanguinous depending on whether there is associated contusion, laceration, or subarachnoid hemorrhage. Death is almost inevitable unless the clot is removed and is due to respiratory arrest.

The surgical treatment is the placement of several burr holes, drainage, identification and ligation of the bleeding vessel. Results are usually quite good.

Tentorial Herniation

If tentorial herniation with crushing of the midbrain occurs before operation, the results are poor. This syndrome consists of bilaterally fixed dilated pupils, failure of vital centres (rising pulse, respiration and temperature) bilateral Babinski signs, and spastic or decerebrate rigidity.

Acute Subdural Hematoma

The lucid intermal may be days to two or three weeks. Blood may originate from *contracoup* lacerations, rupture of veins crossing the subdural space to the venous sinuses, or from a torn sagittal or lateral sinus. Headache, drowsiness, agitation and confusion are frequent symptoms. Signs of papilloedema, slowing of the pulse and respiration are evidence of increasing intracranial pressure. Secondary coma may occur over a few hours to several days. Focal

convulsions, dysphasia, unilateral limb weakness and disturbed reflexes help to lateralize the clot but may be late manifestations. The best guide is the development and depth of unconsciousness. Treatment is by bilateral temporal burr holes and/or subtemporal decompression.

Cerebral Hemorrhage

Acute bleeding of this type is more common in the elderly than in the young and is usually fatal in a few hours or, if the bleeding is slow, in an interval of two to three days from injury. The clinical picture includes deepening coma, hemiplegia, hypereflexia, dilating pupils, bilateral Babinski signs and stertorous irregular breathing. Craniotomy with evacuation has given successful results in a few cases.

COMPLICATIONS

Chronic Subdural Hematoma

The head injury may be trivial especially in the elderly, and is often forgotten. After a period of weeks, headache, giddiness, slowness in thinking, confusion, and, rarely, seizures are the initial symptoms. Disturbances in consciousness may be more profound than focal or lateralizing signs (hemiparesis, aphasia, hemianesthesia or homonymous hemianopsia). If tentorial herniation occurs with compression of the contralateral cerebral peduncles pyramidal tract signs ipsilateral or bilateral to the clot will result. The ipsilateral pupil dilates due to compression and paralysis of the III nerve and may be followed by ptosis. X-ray examination of the skull may be negative or show a shift of the calcified pineal. EEG can be bilaterally abnormal and arteriogram may show displacement of the anterior and middle cerebral arteries. The C.S.F. may be bloody, clear or xanthochromic, the pressure may be elevated, normal or subnormal. The best diagnostic procedure is direct burr hole exploration.

Symptoms in chronic subdural hematoma result from encystment of the clot, hemolysis and disintegration of blood cells causing an increased osmotic pressure. Fluids enter the hematoma resulting in enlargement and consequent compression and displacement of underlying brain tissue.

Acute Subdural Hygroma

Tears in the arachnoid producing a valve-like orifice allow the entrance of blood and C.S.F. resulting in a slowly expanding cystic mass. This spreads diffusely over the hemispheres, causing increased intracranial pressure and signs and symptoms suggestive of subdural hematoma. Exploratory burr holes and drainage serve as the diagnostic and therapeutic tool.

Massive Cerebral Edema

This most commonly follows gross laceration and contusion and gives rise to signs of increasing intracranial pressure. Dehydration therapy should be carried out but not until space-occupying lesions have been ruled out by exploratory burr holes.

Post-Traumatic Meningitis

This most often follows fractures of the paranasal sinuses or petro-mastoid, and septic penetrating wounds. Confusion or semi-coma, fever, nuchal rigidity, Kernig's sign and typical lumbar puncture signs, (increased pressure, turbid fluid, increased protein and WBC count, positive cultures) establishes the diagnosis.

Post-Traumatic Epilepsy

Epilepsy of this type has been reported to follow 20 — 40% of all patients with contusion and laceration to the cerebral cortex. Seizures are focal in nature and generally appear nine months following injury. Anticonvulsant therapy is indicated. Surgical removal of the scar tissue can hardly be recommended for healing is by a process of scar formation.

ACKNOWLEDGMENT — The author wishes to thank Dr. J. Reudy for the helpful criticism of this paper.

MEDICAL SHOCK

L.M. JERRY, 6T1.

This article will deal specifically with "medical shock" in which there is no primary loss of circulating blood volume, with particular reference to shock associated with myocardial infarction, adrenal cortical insufficiency, septicemia, anaphylaxis and pulmonary embolism.

ETIOLOGY AND PATHOGENSIS

Shock may result from a decrease in circulating blood volume (secondary, delayed, prolonged, true or surgical shock). Blood volume may be depleted by hemorrhage, both internal and external; by loss of plasma or whole blood into tissues injured by burns or crushing; and by a deficit in extracellular fluid volume because of a decrease in water intake or because of a loss of water and electrolytes through the G.I. tract,

the kidneys, and the skin (dehydration shock). The essential mechanism is a decrease in inflow load to the heart because of a decrease in circulating blood volume with a resultant fall in cardiac output and, therefore, blood pressure, resulting in failure of the circulation to the periphery.

Shock may result from a primary loss of vascular tone (emotional, immediate, primary, neurogenic shock, fainting, "collapse") resulting from emotional factors (psychogenic syncope); physical insults to the nervous system (sympathectomy, transection of the cord); chemical injury to the central nervous system (hexamethonium and other vasodilators, spinal anesthesia); spinal cord disease (diabetic myelopathy) which incapacitates the postural vasoconstrictor reflex so that syncope occurs on standing; venous pooling in patients with varicose veins or atonic musculature who faint from prolonged motionless standing. The vasovagal attack is the basis of this type of shock in which the above-mentioned etiologic factors bring about vagal stimulation (with bradycardia) and peripheral vasodilatation (skin vessels constrict, hence the pallor) with a resultant fall in peripheral resistance. The decrease in cardiac output and peripheral resistance lowers the blood pressure until peripheral circulatory failure results.

Primary arteriolar and capillary dilatation may rarely result in shock because of the direct action of chemical agents on the vasculature. Examples include anaphylaxis, histamine shock and shock induced by nitrites and local anesthetics.

Finally, some forms of shock tend to be mixed in pathogenesis. In myocardial infarction and acute pancreatitis, the major cause is the decrease in cardiac output and loss of protein rich fluid and electrolytes into the peritoneal cavity, respectively. However, primary loss of vascular tone from obscure reflexes plays a role. Similarly, the shock resulting from the fall in cardiac output from obstruction to the pulmonary artery by an embolus is supplemented by the excitation of similar obscure reflexes. Neurogenic shock precedes surgical shock in any injury or acute catastrophe such as a perforated peptic ulcer. Infection produces shock chiefly by its dilating action on the myocardium and cardiac conduction systems, and failure of medullary control centers contribute to the condition.

CLINICAL RECOGNITION

Sudden development of shock lowers blood pressure before compensation can occur. The resulting cerebral anoxia accounts for the sudden syncope. When shock develops, slowly compensating vasoconstriction has time to keep pace and maintain a blood pressure high enough to ensure adequate cerebral circulation; hence, apathy and lassitude are substituted for fainting. The early diagnosis of incipient shock involves an awareness of the disease conditions which predispose to it. Cold moist or dry, inelastic skin is an early sign. Oliguria (less than 25 ml. per hour on an adequate fluid intake occurs before a fall in blood pressure can be noted. Orthostatic hypotension with a fall in pulse pressure (systolic more affected than diastolic) occurs early; therefore, take blood pressures with the patient sitting in the early stages. The measurement of blood volume with Evan's Blue dye shows a decrease of 20% — 30% from the estimated normal volume before signs and symptoms of shock are observed. Shock of peripheral origin may be confused with cardiogenic shock caused by a painless coronary occlusion or an ectopic tachycardia.

MANAGEMENT

Supportive Measures

Speed, position, airway and analgesia are the essentials, all discussed in more detail in other sections of this Journal.

Parenteral Fluid Therapy

Although replacement of blood volume is the mainstay of treatment for surgical shock, such therapy is not so important in cases of medical shock. The fluids of choice are blood when shock is due to hemorrhage, plasma and saline when burns are responsible, a combination of blood and plasma when there is trauma, and appropriate electrolyte solutions for shock due to dehydration. In addition, make up daily losses by insensible loss and urine volume with glucose solutions. When shock is severe, give blood at the rate of 1 L./hour for the first 3 — 4 hours. Terminate transfusion when signs of shock have disappeared and when a free flow of urine (40 ml. or more per hour) has appeared. Until blood is ready temporize with 500 ml. of saline and/or glucose solution followed by 500 to 1000 ml. of a plasma expander like Dextran or of plasma itself. Once shock is controlled, compensate for insensible water loss with 1 liter of isotonic (6%) glucose solution each 24 hours. Potassium chloride may be added to the infusion if renal function is adequate to compensate for the transfer of potassium to the intracellular compartment when the glucose is stored as glycogen. Oral fluids are given in quantity as soon as tolerated. Over-infusion demonstrates itself by the development of a gallop rhythm, tachypnea, venous distension, increased venous pressure, basal rales, and labored breathing. Stop the infusion or slow it enough to allow intravenous vasopressor therapy if shock is still present, and add Cedilanid to the infusion. Elevate the patient's head and give oxygen.

Vasopressor Drugs

Vasopressor drugs are indicated in the late phases of all severe shock (failure of vasomotor mechanisms because of anoxia) and in shock caused by primary vasodilatation. The best drug is norepinephrine given intravenously (add 5 to 15 ml. of 1:1000 solution to 1 liter of intravenous solution) beginning at 20 to 40 drops per minute and continuing. If the drug enters the subcutaneous space, sloughing of the overlying skin results. Infiltrate the involved tissue with 5 mg. of Rogitine diluted in 20 ml. of water at a rate sufficient to maintain systolic blood pressure. Other vasopressors which may be used in decreasing order of effectiveness are meteraminal (Aramine), Mephentemine (Wyamine), methoxamine (Vasoxyl), phenylephrine (Neo-synephrine). They may be given subcutaneously or intramuscularly in 3 to 15 mg. doses every 15 minutes or in an intravenous infusion (30 to 300 mg. per liter to maintain an adequate blood pressure. An attendant should be present to watch the patient, his blood pressure, and his urine volume.

Evaluation of Therapy

Evaluate pulse, respiration, rectal temperature and blood pressure every 15 to 30 minutes until improvement. Blood Hb., R.B.C. and hematocrit are done serially to evaluate the results of therapy.

SHOCK FOLLOWING MYOCARDIAL INFARCTION

Recognition

The systolic blood pressure falls below 80—90 mm. Hg. in a previously normotensive patient or below 110—120 mm. Hg. in a previously hypertensive patient for 2 hours or longer. The syndrome of myocardial infarction with shock and acute left and right-sided heart failure is seen. Prognosis is grave for 90% of these patients either die or develop such complications as acute tubular necrosis, focal cerebral disorders, gangrene of skin and muscle of the extremities and extension of the myocardial infarct.

Treatment

Commence general antishock measures. Orthopneic position is required in the presence of heart failure. Blood, plasma and salt infusions are harmful since blood volume is normal and the heart has failed. Digitalize the patient rapidly since forward and backward failure are present. Use of the rapid acting Lanstoside C — Cedilanid. The digitalizing dose is 1—2 mg., I.V. in divided doses over 4 to 8 hours, then change to a long acting drug such as 0.1 mg. digitoxin or 9.1 gm. digitalis leaf t.i.d., p.c. for 5 days, then once daily for maintenance. Use a vasopressor such as Levophed, 4—8 mg. in 1000 ml. of 5% glucose in water, immediately at a rate just sufficient to give a pulse pressure of 30 mm. Hg., and a systolic pressure of 90—100 mm. Hg., or higher in a previously hypertensive patient. Stop the infusion if ectopic rhythms begin; if second or third degree A/V block develops, give atropine sulfate 1—2 mg. The vasopressor may be given via a plastic polyethylene catheter passed through a peripheral vein to the region of the superior or inferior vena cava to prevent extravasation or necrosis. In the presence of pulmonary edema or refractory cases use higher concentrations (8 to 12 mg. per 1000 ml.) potentiated with 100 mg. hydrocortisone I.V., or molar lactate to correct acidosis if present. Terminate the infusion gradually after blood pressure has stabilized for more than 6 hours and supplement with intramuscular sympathetic amines every 4 to 6 hours. Administer specific treatment for infarction, failure and their complications as indicated.

SHOCK FOLLOWING MASSIVE PULMONARY EMBOLISM

Recognition

A patient who previously seemed quite well suddenly experiences severe retrosternal pain, becomes cyanotic and intensely dyspneic, and develops vascular collapse. Evidence of phlebothrombosis or thrombophlebitis may be present. Differentiation from coronary occlusion is difficult. Death may occur before treatment can be instituted, or the patient may recover slowly from a state of profound shock under appropriate management.

Treatment

Therapy must be undertaken while the diagnosis is still presumptive if it is to be successful. The treatment is like that of shock in association with coronary occlusion. Use Demerol rather than morphine (which is said to cause a greater respiratory depression). Papaverine, 30—60 mg. and atropine, 1—2 mg. I.V. may be given slowly and repeated every 3 to 4 hours in an attempt to overcome the general pulmonary arteriolar spasm that reflexly occurs. Institute immediate prophylactic and specific management for venous thrombosis (anti-coagulants, venous ligation, etc.). Watch for secondary infection and for a pleural effusion which is removed by thoracentesis if it embarasses respiration.

SHOCK ACCOMPANYING ANAPHYLAXIS

Recognition

Following the parenteral administration of foreign sera, antibiotics or drugs, the patient suddenly experiences signs and symptoms which may include any or all of apprehension, paresthesias, generalized urticaria or edema, choking sensation, cyanosis, wheezing, cough, incontinence, shock, fever pupillary dilatation, loss of

consciousness, and convulsions. Death may occur within 5 to 10 minutes. A history of previous exposure to the drug may be lacking.

Treatment

Apply a tourniquet on the arm or leg proximal to the site of injection. A tracheotomy may be necessary for laryngeal edema. If circulatory collapse is present give vasopressors: epinephrine I.V., 0.2 ml. of 1:1000 solution (or subcutaneously if venous collapse is present and follow with an I.V. Levophed drip (0.2 or 4 ml. in 1000 ml. of 5% glucose in water). Place the patient in Trendelenburg position. If respiratory distress is marked however, give the patient oxygen and sit him up. Now give hydrocortisone (100 mg. of Solu-Cortef) plus an antihistamine (Benadryl, 50 mg.) I.V. If wheezing is marked give Adrenalin (epinephrine) 1:100 or Isaprel (isoproterenol) 1:200 in a nebulizer. Oxygen is given by intermittent positive pressure at 6 liters per minute. Mild wheezing will respond to aminophylline 0.5 gm. I.V. Do not give narcotics, but terror may be relieved by the use of phenobarbital.

SHOCK ASSOCIATED WITH SEPTICEMIA

Recognition

Early and accurate diagnosis of the causative organism, its primary focus, and its sensitivity to antimicrobial agents is essential. Indications of blood stream invasion include failure of expected improvement; further increase of temperature, or septic manifestations (rigors, shock, prostration, icterus, purpura, hyperpyexia, delirium, coma, acute renal failure). Take blood cultures for aerobic and anaerobic organisms repeatedly and frequently. Until these results are available to dictate specific therapy, empiric treatment is employed based upon etiologic probabilities. Hemolytic streptococci invade the blood stream from a wound cellulitis, middle ear infection, pelvic phlebitis, and peritonsillar abscess. Staphylococcal septicemia may follow a carbuncle, osteomyelitis, wound infection, intravenous cut-down, or heart operation. Gram-negative bacillary septicemia (Pseudomonas, Proteus, Salmonella) may arise from the gastrointestinal tract, the biliary tree, the urinary tract and open wounds. Septicemia in patients treated with tetracylines may be caused by Pseudomonas, Proteus or Staphylococcus; with chloramphenicol it may be Pseudomonas or a Coliform. Staphylococcus and Gram-negative Coliforms invade the blood stream of patients on steroid or bone marrow suppressant therapy. Pseudomonas is usually present in infected, transfused blood. Shock also may be associated with Cl. welchii infections, H. influenzae infections (Waterhouse — Friderichsen syndrome), viral hemorrhagic fever, epidemic and murine typhus, and Rocky Mountain Spotted Fever.

Treatment

The choice of drug is guided by sensitivities and past experience. A combination of bactericidal agents is given in maximum tolerated doses by a parenteral route. If allergy to penicillin exists, combine streptomycin with one of erythromycin, novobiocin or bacitracin. Duration of therapy is a matter of experience and is based on observations of the patient's well-being, interest in his surroundings, appetite, mental alertness, evidence of recession of primary or metastatic bacterial inflammations. Negative blood culture and fall in temperature may occur before the infection is eradicated.

Blood transfusions are given for anemia and hypoproteinemia. Freshly drawn blood is given for thrombocytopenia and agranulocytosis. Gamma globulin is given for agammaglobulinemia to supply natural antibodies against Gram-negative organisms and for infections refractory to antimicrobial therapy alone. Antishock, supportive and symptomatic measures are applied as indicated. Fluids and electrolytes are administered for the treatment of dehydration (with oliguria, uremia and acidosis) and to supply daily fluid requirements. Urinary output is maintained at 1500 ml./day. Severe hypotension may require the use of intravenous vasopressors and, if necessary, hydrocortisone. Treat congestive heart failure should it develop. Surgical incision and drainage of pus pockets must be done to allow access for cellular, humoral, and chemotherapeutic elements in the blood stream.

SHOCK ASSOCIATED WITH ACUTE ADRENAL INSUFFICIENCY

Recognition

Adrenal crisis is an acute, potentially fatal intensification of adrenal insufficiency and occurs in several ways. Addisonian patients develop a crisis when their hormone requirements rise and are unmet during conditions of significant stress such as infection, trauma, surgery, gastrointestinal upsets, etc. In previously untreated patients, the clinical picture is an intensification of Addison's disease with marked anorexia, muscular weakness, intractable nausea and vomiting, and an increase in pigmentation. Abdominal pain may be severe. Fever may be extreme or subnormal even in the presence of infection. Lethargy developed into somnolence and coma. Vascular shock and dehydration supervene. Laboratory studies show hyponatremia, hyperpotassemia, azotemia and occasionally hypoglycemia. However, in patients previously treated by hormones, severe dehydration, shock and serum electrolyte abnormalities do not occur until preterminally.

Adrenal crisis may occur as a result of adrenal hemorrhage from birth trauma (breech present-

ation), hemorrhagic disease of the newborn, advanced hypertension and arteriosclerosis, during pregnancy, following idiopathic adrenal vein thrombosis, during convulsions in epilepsy or electroshock therapy, with excessive anticoagulant therapy, and after trauma. It is seen associated with Waterhouse-Friderichsen syndrome due to meningococcus, pneumococcus, staphylococcus, and H. influenzae. The onset is explosive with chills, violent headaches, vertigo, vomiting and prostration. A petechial rash appears on the skin and mucous membranes and progresses rapidly to a confluent, extensive purpura. Temperature may be subnormal or extremely elevated; meningismus may be present; leukocytosis occurs; serum electrolytes are normal. In a few hours shock and cyanosis develop, the entire course of the illness lasting only 6 to 48 hours.

Crisis also may occur in association with the adrenogenital syndrome in children, following adrenal or pituitary surgery, and following the application of exogenous stress to patients in whom pituitary-adrenal suppression has occurred following steroid therapy.

Treatment

Therapy involves (1) replacement of the absent steroids, (2) reversal of the metabolic abnormalities, and, (3) treatment of the precipitating cause (usually infection). Speed is essential; overtreat rather than undertreat.

Begin an intravenous infusion of 500 to 1000 ml. of 5% glucose in normal saline containing 100 to 150 mg. of Solu-Cortef. Infuse the first 250 mg. rapidly and the remainder slowly over 4 to 8 hours. An additional 50 to 100 mg. of hydrocortisone may be added to subsequent infusions during the first 24 hours if necessary (usually 12 hours). Give 100 mg. of cortisone acetate I.M., 25 mg. in 4 different sites as well. Follow with single injections of cortisone, 25 — 50 mg., I.M. every 6 hours. and gradually lengthen the intervals of administration to 25 mg. every 8 hours. When vomiting stops, oral fluids and soft foods may be given as may oral cortisone, 12.5 to 25 mg. every 6 hours. Reduce the dose gradually to maintenance levels of 50 — 75 mg. per 24 hours whence additional salt-retaining effect is required and concomitant desoxycorticosterone or fluorohydrocortisone is used.

Institute antishock therapy; hypotension is corrected by giving Neo-Synephrine or levophed as continuous I.V. infusions with the steroid.

Correct dehydration and electrolyte loss, and supply fluids for maintenance of normal body losses until oral fluids can be taken. With a history of prolonged or severe vomiting or diarrhea, or if hyponatremia exists, give 1 to 2 liters of 5% glucose in saline solution in addition to the first infusion containing the steroid and vasopressor. However, if serum sodium is normal and the crisis has developed rapidly, give additional fluids as 5% dextrose in water.

If infection is present, obtain appropriate specimens for culture and bacterial sensitivity studies to guide intensive antibiotic therapy; meanwhile, give aqueous penicillin, 300,000 units every 6 hours.

Be prepared to treat complications which may include overhydration (cerebral or pulmonary edema), hypokalemia, hyperpyrexia, complications of adrenal steroid therapy (psychosis, etc.).

ACKNOWLEDGMENT — I wish to thank Professor K.J.R. Wightman for helpful suggestions made in the preparation of this paper.

CARDIAC EMERGENCIES

P.A. KORHONEN, 6T2.

Myocardial infarction is one of the commonest emergencies that faces the practising physician. Since cardiac arrhythmias often arise in myocardial infarction as complications, prompt recognition of the complications are often life-saving, especially in ventricular fibrillation and heart arrest.

METHOD OF EXAMINATION

An *orderly* examination is of prime importance in the clinical diagnosis of cardiac arrhythmias.

(1) With the patient lying flat, the jugular bulbs and external jugular veins are observed for pulsations. With practice, the "a" (atrial contraction) "c" (ventricular contraction), and "v" (ventricular filling waves may be identified.

(2) The sequence of beats in the radial pulse is analyzed.

(3) The rhythm is analyzed by auscultation,

correlating it with radial and carotid pulses.
(4) The apex and radial rates are counted simultaneously for one minute to estimate the pulse deficit.
(5) An electrocardiogram is taken and studied. If careful clinical examinations are made and then correlated with electrocardiograms whenever possible, skill can be acquired which permits proceeding without them when they are not available.

ATRIAL PAROXYSMAL TACHYCARDIA

Atrial paroxysmal tachycardia is a rapid, regular beating of the heart, caused by stimuli arising from an ectopic focus in the atria and characterized by sudded onset and sudden offset.
Etiology: The cause may be either idiopathic or associated with excessive alcohol intake, excessive smoking, acute infections, mitral stenosis, throtoxicosis, myocardial infarction or Wolf-Parkinson-White syndrome.
Diagnosis: If the paroxysm is brief, symptoms may be absent. Some patients feel a fluttering of the heart starting suddenly, palpitation, throbbing and fulness of the neck vessels. They may also experience weakness, faintness, and shortness of breath. If the paroxysm is prolonged, heart failure may occur. The heart rate is usually about 180 (150-250) and regular, not influenced by rest, posture or exercise. The pulsations in the jugular veins are rapid, and "c" waves may be identified. The pulse is rapid and of small volume. If carotid sinus pressure terminates the attack, this supports the diagnosis. In the electrocardiogram, abnormal P waves are detected with the PR interval exceeding 0.10 sec. and usually associated with a supraventricular form of QRS complex.
Treatment: At first, mechanical methods to induce vagal effects should be tried. These include holding the breath as long as possible, expiring against the closed glottis, inspiring after closing the glottis, bending forward, mechanical induction of nausea, or carotid sinus pressure. For the latter, the patient should be recumbent and 1 ml. of 1/1000 adrenaline for hypodermic use must be ready should asystole persist too long. The right, then the left, and then both carotid sinuses are massaged gently for five to ten seconds each time.

If the mechanical methods are ineffective, drug therapy can be tried. The most effective drug is Digitalis. With the patient in bed, digitalis leaf is given as follows: 0.4 gms. every six hours for three doses, and then 0.2 gms. every twelve hours for the next two days or until digitalization is complete. Other digitalis preparations such as Digoxin, Lanatoside C, or Ouabain may be used in appropriate dosages.

Prostigmine (Neostigmine) 0.5 to 1 mg. subcutaneously effectually stimulates the vagal mechanism but should not be used for asthmatic patients. Quinidine Sulphate may be given in five daily doses every two hours starting at 0.2 gms. The dose is increased 0.2 gms. each day until the dose is 0.8 gms., providing none of the contraindications for the drug are present and the tachycardia has not reverted. Whenever doubt exists between atrial and ventricular tachycardia, quinidine is the drug of choice. Vasopressor drugs may be used to elevate the blood pressure to 160 mm. Hg. inside the carotid sinus. This sometimes effectively stops the tachycardia by vagal reflex action.

VENTRICULAR PAROXYSMAL TACHYCARDIA

This is a regular rhythm with a rapid rate of 140-160 per minute caused by stimuli originating from an irritable focus in one of the ventricles and characterized by a sudden onset. It may be considered as a rapid succession of premature contractions.
Etiology: This type is much less common than the atrial type and is more serious. It can complicate arteriosclerotic heart disease with myocardial infarction, acute rheumatic fever, or be caused by toxic doses of digitalis or quinidine.
Diagnosis: The patient experiences symptoms similar to those of atrial paroxysmal tachycardia. The rate is slightly slower (150-180), and the rhythm is more irregular than the atrial type. Frequently "a" waves may be identified in the jugular vessels. Carotid sinus pressure is ineffectual. The electrocardiogram confirms the diagnosis. The complexes have the form of ventricular premature contractions. The pattern is usually characteristic but at times it may be quite altered and difficult for the inexperienced observer to recognize.
Treatment: The mechanical measures used in the atrial type are entirely ineffective therefore drugs only must be used.

Oral quinidine sulfate is the drug of choice if the situation is not too urgent and if the attack is not due to digitalis or quinidine intoxication. 0.4 gms. every two hours for five doses a day, increased by 0.2 gms each succeeding day, is given until the attack is terminated or toxic effects occur. On return to sinus rhythm, quinidine may be kept at a maintenance dose of 0.2-0.3 gms. every four hours for a few days.

In an overwhelming emergency two drugs are available for intravenous administration. Quinidine gluconate 0.8 gms. in a 10 cc. vial is diluted to 20 cc. with normal saline and administered at a rate of 1 cc. per minute. It is wise to observe a continuous ECG while the drug is

being injected. The drug is then discontinued on restoration to normal sinus rhythm. Procaine amide (Pronestyl) is used if quinidine is ineffective or if the patient is known to be intolerant to the drug. It is given by slow I.V. drip in 5% glucose solution. Frequent or continuous ECG monitoring is advisable and the drug is stopped on restoration of sinus rhythm. The blood pressure should be observed frequently since Pronestyl produces significant hypotension, and Levophed should be available for its control.

ATRIAL FIBRILLATION

Rapid fibrillatory waves take the place of normal atrial contractions and the ventricles respond erratically. The atrial rate is 350-600 per minute, and the ventricular rate is 100-150 per minute.

Etioloty: Atrial fibrillation is the most common rhythm requiring therapy, and can occur with or without organic heart disease. It is associated with excessive intake of alcohol, overeating, and oversmoking. It may complicate surgical operations, acute rheumatic fever, pneumonia, acute coronary occlusion, pulmonary infarction, endocarditis, diphtheria or thyrotoxicosis.

Diagnosis: The patients complain of irregular beating of the heart, palpitation, fullness and fluttering of the neck veins. Apprehension, dizziness or faintness may be present. On auscultation, the rhythm is entirely irregular. The pulse is irregular and of variable volume. Simultaneous auscultation and palpation of the pulse reveals a notable pulse deficit. With moderate exercise the ventricular rate and pulse deficit increase. Carotid sinus pressure retards the ventricules only transiently. The electrocardiogram is characterized by the absence of normal P waves, the presence of fibrillation (f) waves and irregular sequence of the QRS complexes.

Treatment: If the ventricular rate is slow and accelerates only moderately on exercise and if the patient has no symptoms, special treatment is not indicated. However, if the rate is rapid, especially if the patient has heart disease, bed rest is necessary. Providing digitalis has not been given in three weeks, digitalize the patient with oral digitalis leaf as prescribed previously. If the patient is partly digitalized, 0.2-0.3 gms., 2 or 3 times a day may be given. Cardiac rate and pulse deficit are determined whereupon successive doses are withheld if nausea or vomiting occur, or if the ventricular rate falls below 60-70 per minute. Having reduced the rate, it is maintained at this level by ration amounts, the average being 0.2 gms. per day using the rate as a guide.

Reversion to normal rhythm by quinidine may be providing the patient is not too old, if the fibrillation has not been present for too long, if the heart is not enlarged and if there is no congestive heart failure. The ventricular rate is first reduced with digitalis, which is then discontinued and a test made for quinidine idiosyncrasy with 0.2 gms. orally. Three hours later 0.4 gms. is given and 0.4 gms. every two hours for a total of five doses. If the arrhythmia persists and the patient is free from the toxic effects of quinidine, the next day 0.6 gms. is given every two hours for five doses. Then, if necessary, 0.8 gms. is given q. 2 h. x 5 on the third day and the same dose is repeated on the fourth day if the arrhythmia persists. If after four successive days of therapy as outlined above, the arrhythmia persists, it is best to abandon quinidine medication and rely on digitalis to maintain an apical rate between 60-70 per minute. Another attempt at conversion may be made after a few weeks.

ATRIAL FLUTTER

In this rhythm there is a rapid regular atrial action at a rate of 250-350 per minute and the ventricle responds usually to every other beat (2:1 block). Sometimes the response is slower, eg. 3:1 or 4:1 and occasionally the degree of heart block varies so that the rhythm is irregular.

Etiology: Atrial flutter is commonly associated with heart disease but is less common than atrial fibrillation. The etiology is approximately the same.

Diagnosis: The symptoms are similar to atrial fibrillation. The ventricular rate is usually rapid (150-160) and not influenced by holding the breath. Occasionally, rapid jugular venous "a" waves (300 per minute) may be detected. Carotid sinus pressure may reduce the rate by half temporarily but sinus rhythm is not restored. The ventricular rate is usually 150-165 per minute and regular. However, when the A-V block changes, the ventricular beat may seem irregular during the transitional period. The ventricular rate decreases as the block increases. On the electrocardiogram, the detection of regularly spaced "sawtoothed" flutter waves (designated as "F" waves) establishes the diagnosis.

Treatment: During treatment the patient should be in bed. Digitalis is the most effective drug. Oral digitalization may be accomplished by giving digitalis leaf in divided doses over two or three days. As it becomes effective the ventricular rate falls as the block increases. During digitalization, the rhythm may become normal, the block may change, or it may convert to fibrillation. After the onset of fibrillation, normal rhythm may follow. If normal rhythm is not achieved by digitalis, or if the rhythm has changed to fibrillation which persists, quinidine

therapy could be tried. At this stage of therapy some authors advise stopping the digitalis, others continue digitalis therapy should 1:1 conduction arise due to the quinidine. Quinidine sulphate 0.4 — 0.8 gms. q. 2 h. x 5 should be given as described for atrial fibrillation. If quinidine is effective, maintenance amounts are given for a few days.

Procaine Amide in large amounts occasionally restores normal rhythm in chronic atrial flutter but is more effective in the paroxysmal type. A dangerous side effect is the onset of 1:1 conduction.

Recurrent flutter may be prevented by digitalization followed by ration doses.

VENTRICULAR FIBRILLATION

In this rhythm the co-ordinated contractions of the ventricles are replaced by rapid irregular twitches of the ventricular musculature. Its persistence is incompatible with life since the twitchings are inefficient in propelling blood.

Etiology: Ventricular fibrillation may be caused by electrocution, chloroform or cyclopropane anesthesia, or acute coronary occlusion. It may follow paroxysmal ventricular tachycardia or toxic doses of digitalis or quinidine.

Diagnosis: The patient loses consciousness. There is an absence of pulse, the blood pressure drops to zero and heart sounds become inaudible. At the onset of fibrillation, an ECG would show five or six deflections similar to those produced by ventricular extrasystoles, but occurring very close together. Then the deflections become lower in height, and more erratic in frequency and shape. Within a short time, variable from patient to patient, no electrical activity is recorded as the fibrillation is followed by ventricular standstill.

Treatment: This is largely preventive and consists of using every precaution when administrating drugs that induce fibrillation. The use of quinidine or procaine amide to prevent ventricular fibrillation after acute coronary occlusions should be considered. Before cardiac operations, the heart should be made more resistance to external stimuli by administration of quinidine or procaine amide.

In ideal conditions and with an electrical defibrillator immediately available, ventricular fibrillation may be stopped by an electric countershock of 250 volts, 60 cycles for 0.15 seconds applied through the chest wall with large electrodes. After the heart is stopped by this method it may be necessary to apply an external pacemaker to keep the heart pumping until the normal pacemaker takes over.

If an electrical defibrillator is not immediately available, it will be necessary to open the chest and massage the heart until the heart can be defibrillated by an electric shock of 110 volts, 2-3 amps., 60 cycles. If this is not done within three minutes, death may ensue.

CARDIAC ARREST

Cardiac arrest occurs when the heart suddenly stops beating and fails to restart.

Etiology: Cardiac arrest occurs most commonly as a complication of cardiac surgery and less frequently in general surgery. It may also occur in arteriosclerotic heart disease with or without myocardial infarction, or in any acute infection. Sometimes it may occur without any apparent reason.

Diagnosis: The patient loses consciousness, his blood pressure falls to zero, ther is no pulse, the heart sounds are absent, and no electrical activity is recorded in the ECG.

Treatment: This is a matter of utmost urgency as the blood flow to the heart and brain must be restored in three or four minutes. Slap the precordium forcefully or punch it vigorously with the side of the fist. If standstill persists, epinephrine 0.1-0.5 ml. of 1/1000 solution may be injected directly into the heart chamber. When an electrical cardiac pacemaker is immediately available, some specialists advocate external electrical stimulation. Others recommend external manipulation by heavy compression of the lower sternum and the xiphisternum which can produce an effect equivalent to direct massage of the heart. This can be applied in selected adult cases and particularly in children. If all other measures fail, thoracotomy and direct cardiac massage may be undertaken as a last resort. The method chosen should be determined according to the equipment available.

ACUTE MYOCARDIAL INFARCTION

Acute myocardial infarction is the clinical syndrome characterized by prolonged substernal pain, a fall in blood pressure and other manifestations of shock, accompanied by characteristic progressive electrocardiographic changes, fever, leukocytosis, an increased sedimentation rate and increased serum glutamic oxaloacetic transaminase level (SGOT).

Etiology: The principal cause is the occlusion of a coronary artery by thrombosis, subintimal hemorrhage, rupture of an atheromatous abcess, or by an embolism. Other causes are arteritis due to syphilis, rheumatic fever, systemic infections or thromboangitis obliterans.

Diagnosis: The onset of myocardial infarction is accompanied by the appearance of severe persistent substernal pain. The patients may describe it as sharp, agonizing, vicelike, tearing or burning. The pain is usually referred to the upper or middle third of the sternum, but may beneath the lower third, over the precordium,

or in the epigastrium. Radiation is usually to the left arm and shoulder, at times to the right arm, to both sides of the neck or more rarely, down to the abdomen. In contrast to the invariable immobility with angina pectoris, some persons become restless and may even pace around the room. The pain is distinctively different from that of angina pectoris. It is different in quality, in its greater severity and duration, and in its failure to respond to nitroglycerine.

With the onset of the attack the patient becomes pale, is often in cold sweat, and is dyspneic, weak and apprehensive. The blood pressure may be at shock levels. The pulse may not be detectable at the wrist and may be only barely palpable at the carotids. The pulse rate is above 100 and the heart sounds are weak. Gallop rhythm, atrial fibrillation, atrial and ventricular tachycardia may be present. On auscultation one may hear a pericardial friction rub or moist rales.

Fever and leukocytosis are usually observed in the first twenty-four hours, the magnitude being proportional to the severity and extent of the infarction. The fever is usually 101° F. although it may go up to 105°. Leukocytosis is usually from 12,000 to 15,000 per cu. mm. occasionally rising as high as 25,000. In the next three to four days, the erythrocyte sedimentation rate (ESR) becomes elevated. The SGOT elevation is first detected about twelve hours after the onset, is at its peak three or four days later and reverts to normal (i.e. below 40 units) in one week. The degree and duration reflect the extent of the cardiac damage to a certain degree.

In the electrocardiogram, there are characteristic QRS changes, ST segment displacements and T wave inversions. The ST segment is elevated above the isoelectric line and bowed convexly upwards. The T waves become inverted. If the infarct extends right through the ventricular wall, abnormal Q waves also appear. These changes most frequently occur in the chest leads recorded over the affected area and may be reflected in the limb leads. It is important to obtain serial readings since the changes may not appear for several days.

Control of Pain and Apprehension: Promptly administer 15 to 30 mgms. morphine sulphate subcutaneously. A second dose is given if the pain is not relieved in 30 minutes. Demerol 100 mgms. I.M. may be used instead of morphine. When morphine is used, it should be accompanied by 0.6 - 1.0 mgms. atropine subcutaneously to reduce vagal stimulation. For mild pain, one gives codeine 30 mgms. subcutaneously. During the acute illness, sublingual nitroglycerine 0.45 - 0.64 mgms. will often bring relief. Sufficient sedation should be given to reduce anxiety and restlessness with 15 - 30 mgms. phenobarbital. The extremely anxious patient should be tranquilized with 0.5 - 1.0 mgms. Reserpine or 0.4 to 1.2 gms Equanil orally.

Control of Cardiac Mechanism: Disturbances of the cardiac mechanism must be anticipated routinely. During the first few days and particularly if arrhythmias are present, the heart should be frequently checked with an ECG. The following disturbances should be watched for and treated as outlined in the first part of this article. Premature ventricular beats, persistent ventricular tachycardia, paroxysmal ventricular tachycardia, atrial tachycardia, atrial fibrillation and flutter, heart block, (a) SA block, (b) AV block.

Control of Blood Pressure: In hypotension without shock, mild pressor drugs are indicated to prevent secondary myocardial depression. Intravenous Wyamine 15-60 mgms., Aramine 3-10 mgms., or Ephedrine 15 mgms. may be used, followed in one half to two hours by equal intramuscular doses.

In hypotension with shock, the value of immediate vigorous pressor treatment has been established beyond doubt. Intravenous Wyamine, Aramine, or NeoSynephrine is given while a diluted solution of norepinephrine 4-8 mgms. in 1000 cc. 5% glucose in distilled water is being prepared. Next, a plastic polyethylene catheter is inserted through a peripheral vein to the region of the superior vena cava to prevent extravasation and necrosis. The rate of norepinephrine is adjusted to a flow of 1-2 micrograms per minute to maintain the systolic pressure at 90 to 100 mm. Hg. or higher. One terminates the infusion gradually after the blood pressure has been re-established for more than six hours, and intramuscular sympathetic amides are given as supplement every 4 - 6 hours.

Control of Myocardial Muscle Failure: Acute pulmonary edema requires immediate treatment. One gives 100% oxygen by positive pressure of 3-4 cm. Hg. Rotating venous tourniquets are applied at the proximal ends of the extremities using blood pressure cuffs set at 45-50 mm. Hg. Intravenous digitalization is performed by giving 1.6 mgms. Lanatoside C in divided doses.

Paroxysmal nocturnal dyspnea or congestive failure requires less urgent therapy: full oral digitalization, salt restriction, fluid restriction, elevation of the head of the bed, and mercurial diuretics.

Control of Thromboembolism: Providing there are no contraindications, antigoagulants should

oral CHLOROMYCETIN Kapseals*—the opaque white capsules with grey band—for unvarying potency, purity, and efficacy from dose to dose • CHLOROMYCETIN Palmitate—for children and adults who prefer a liquid medication—an easy-to-give, easy-to-take custard-flavoured suspension. **parenteral** CHLOROMYCETIN Succinate—so versatile it can be given intramuscularly, intravenously, or subcutaneously.

CHLOROMY

ophthalmic CHLOROMYCETIN Ophthalmic Ointment and CHLOROMYCETIN Ophthalmic Solution for notable efficacy...ready penetration into ocular tissues. **topical** CHLOROMYCETIN Cream, 1% for rapid clearing of superficial skin infections and surgical wound infections • CHLOROMYCETIN Topical, 10% solution, for excellent results in infections of grafts, surface infections, and varicose ulcers.

Since its introduction early in 1949, the unusually broad range of antibacterial action of CHLOROMYCETIN, its purity, outstanding tissue diffusibility, and low incidence of gastrointestinal side effects have combined to make this Parke-Davis antibiotic an agent of choice throughout the years[1-14] in a wide variety of systemic and localized infections.

The numerous dosage forms of CHLOROMYCETIN—designed to meet virtually every type of clinical application—offer the physician the added convenience of flexibility of dosage and administration according to the requirements of the patient.

CETIN*

AN APPROPRIATE DOSAGE FORM FOR EVERY TYPE OF CLINICAL APPLICATION

(chloramphenicol, Parke-Davis)

Chloramphenicol is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy. Medical literature available on request.

References: (1) Ehrlich, J., *et al.: Science* **106**:417, 1947. (2) Woodward, T. E., *et al.: Ann. Int. Med.* **29**:131, 1948. (3) Smadel, J. E.: *Am. J. Med.* **7**:671, 1949. (4) Parker, R. T., *et al.: J.A.M.A.* **143**:7, 1950. (5) Lewis, R. S., & Gray, J. D.: *Brit. M. J.* **2**:939, 1951. (6) Trice, E. R., & Shafer, J. C.: *J.A.M.A.* **149**:1469, 1952. (7) Robinson, H. M., Jr., *et al.: Bull. School Med. Univ. Maryland* **38**:109, 1953. (8) Roper, K. L.: *Indust. Med.* **23**:50, 1954. (9) Costner, A. N.: *South. M. J.* **48**:1192, 1955. (10) Deacon, W. E., *et al.: Antibiotic Med.* **2**:143, 1956. (11) Josephson, J. E., & Butler, R. W.: *Canad. M.A.J.* **77**:567, 1957. (12) Blair, J. E., & Carr, M.: *J.A.M.A.* **166**:1192, 1958. (13) Goodier, T. E. W., & Perry, W. R.: *Lancet* **1**:356, 1959. (14) Rebhan, A. W., & Edwards, H. E.: *Canad. M.A.J.* **82**:513, 1960.

PARKE-DAVIS

Parke, Davis & Company, Ltd., Montreal 9, P. Q.

*REGISTERED TRADEMARK

CP-56160

be given in all cases, whether the patients are good or bad risks. For the first 24-48 hours, the patient is heparinized until Dicoumarin therapy becomes effective. One gives 50 to 100 mgms. of heparin I.V. three times during the day and twice during the night, while at the same time Dicoumarol 300 mgms. is given the first day. Subsequent doses are gauged daily after the daily prothrombin time is available. 200 mgms. is usually given the second day, and 50 to 100 mgms. each subsequent day to maintain the prothrombin time at about 30 seconds When the prothrombin time is 35 seconds or over, the dose is omitted.

Myocardial infarction is much more common than the arrhythmias yet it is important to understand the arrhythmias fully since they so often complicate this condition.

ACUTE PULMONARY EDEMA

B. FOGEL, B.A. 6T1.

Acute pulmonary edema is frequently encountered as a medical emergency. Treatment must be immediate regardless of how drastic the patient's condition may appear in order to prevent death.

ETIOLOGY AND PATHOPHYSIOLOGY

Blood flow through the pulmonary vascular bed is approximately 5L/min., pulmonary capillary pressure being about 5 to 8 mm Hg. above intrathoracic pressure levels. Since the osmotic pressure in blood is approximately 25 to 30 mm Hg., the pulmonary capillary pressure must be significantly elevated before acute pulmonary edema becomes clinically manifest.

From the clinical standpoint, three basic mechansisms are chiefly responsible for the development of acute pulmonary edema.

a) *Cardiac-circulatory:* clinically the most important, due primarily to an increase in the pulmonary capillary pressure, secondary to an acute failure of the left ventricle. Acute left ventricular failure is the commonest cause of pulmonary edema and is usually associated with hypertensive cardiovascular disease, arteriosclerotic heart disease, valvular heart disease (mitral or aortic) or in association with myocardial infarction. While it is known that increased pulmonary capillary pressure due to pulmonary congestion in heart disease favours pulmonary edema, body water balance is equally an important factor. The increase in reabsorption of fluids at night is a factor which may bring on paroxysmal nocturnal dyspnea and pulmonary edema in cardiac patients.

b) *Alveolar:* where there is direct damage to the alveolar-capillary membrane. Chemical injury is another cause of acute pulmonary edema. Some of the more common irritants encountered in industry include: chlorine, bromine, ammonia, sulfur dioxide, fumes of strong acids and volatile organic solvents. Trauma, involving the alveolar capillary membrane may also produce acute pulmonary edema. Among the causes are lung injuries from various causes including blast and burn from inhalation of hot gases.

c) *Tracheobronchial:* where there is inadequate drainage of pulmonary airways and subsequent accumulation of intra-alveolar exudate. It is obvious that this is not a frequent cause of acute pulmonary edema as seen in the emergency department and therefore will not be considered.

SYMPTOMS AND SIGNS

Acute pulmonary edema presents with fairly characteristic symptoms and signs. Dyspnea and orthopnea occur early. In a fully established case, the patient is usually found sitting upright, gasping for breath, with the mouth flecked with pink frothy sputum. Pallor, sweating and cyanosis are usually present. Coughing is usually a constant feature. Rales, bubbling in character, wheezing (cardiac asthma) and sonorous rhonchi are heard in the chest. The pulse may be rapid and there may be a drop in blood pressure.

Ambulant patients with severe mitral stenosis are often thrown into pulmonary edema after sudden exertion. Cough, increased dyspnea and frothy pinkish sputum with transient moist rales are frequent findings. Hypertensive patients characteristically develop a severe form of pulmonary edema characterized by anxiety, cough and pressure in the chest, orthopnea with laboured dyspnea. Cyanosis, moist rales, pallor, sweating and an increase in pulse and blood pressure are found on examination.

DIAGNOSIS

The clinical findings plus a history of pulmonary or cardiac disease, exposure to irritant gases or fumes, or other etiological factors are usually sufficient to make a diagnosis of acute

pulmonary edema. Conditions with which this might be confused are bronchopneumonia and bronchial asthma.

TREATMENT

Simple measures employed early will do much to ease the patient's distress. The patient is usually placed in the orthopneic position with the legs dependant. If fully conscious, he is most accomodating to this measure.

Morphine Sulphate should be given immediately. This may be given intravenously in a dose of 1/4 gr. Depending on the age and build of the patient, 1/6 gr. may also be given I.M. Morphine not only relieves the dyspnea by allaying the patient's apprehension, it also reduces the sensitivity of the respiratory centre to the afferent impulses of the Hering-Breuer reflex. Care must be taken in advanced pulmonary disease such as pulmonary emphysema where Demerol is better tolerated. In acute pulmonary edema due to alveolar irritation from irritant gases or fumes, morphine and other sedatives are usually contraindicated inasmuch as they depress respiration and inhibit the cough reflex.

Oxygen is usually available in most emergency departments. If this is not the case, oxygen must be administered on subsequent arrival to the ward. Although any method is satisfactory, it is advisable to avoid the oxygen mask as this only adds to the patient's apprehension. The oxygen tent is the preferable mode of administration. The volume of oxygen required by patients with pulmonary edema may be initially as high as 15 to 30 L/min. (100%). If a favourable response occurs, positive pressure oxygen inhalation may be continued until the acute symptoms subside, after which it may be tentatively discontinued to be resumed on subsequent evidence of further cyanosis, coughing and dyspnea.

Aminophylline is frequently used and this tends to relieve any existing bronchospasm in many instances. The usual dosage is 7 1/2 gr. I.V. given over a 7 1/2 — 10 minute period. Aminophylline also supposedly stimulates the myocardium and increases GFR. Its one disadvantage is that it may produce vomiting. This may be avoided by giving it slowly and carefully.

Digitalization: If the pulmonary edema is on a cardiac basis, rapid digitalization by one of the cardiac glycosides is mandatory. Intravenous digoxin, 0.5 mg. stat and 0.25 mg. q2h to a dose of 1.5 mg. affords an adequate form of therapy.

Diuretics: The use of diuretics are only adjuvants to therapy, the benefit being somewhat delayed. Thiomerin, 2cc. S.C. may be given *stat.*

Tourniquets: The application of venous tourniquets is usually unnecessary. However they may be applied to the patient's legs, and, if necessary, to the arms, in order to pool the blood and thus prevent blood from returning to the heart. This, in conjunction with the above specific therapy, often brings about dramatic relief. Ischemia is to be avoided by the prompt release of the tourniquet at definite intervals of 15 minutes.

Venesection: Venesection is always described as a mode of therapy in the treatment of acute pulmonary edema. Adequate recovery is usually obtainable without resorting to such drastic measures. However if conservative therapy fails, one should perform venesection. Three to five hundred cc. of blood should be removed, the amount being estimated by the relief obtained by the patient.

SUMMARY

A cursory explanation of the etiology and pathophysiology of acute pulmonary edema is outlined. Symptomatology, diagnosis and treatment are considered, stress being placed primarily on the latter. Treatment by the "cardiac cocktail" i.e. morphine, aminophylline, digoxin and oxygen is outlined. Reference is also made to the use of venous tourniquets and venesection.

STATUS ASTHMATICUS

G.J. DONSKY, 6T1.

Asthma is a condition characterized by wheezing, expiratory dyspnea, and cough occurring spasmodically, particularly after exposure to air-borne antigens. Although allergy is the most common cause the condition may be precipitated by a bacterial infection. Status asthmaticus may beither an acute severe attack or intractable asthma which may persist for hours, days, or weeks with little relief even during sleep, resulting in great exhaustion to the patient. In Canada, there are approximately 650 deaths per year from asthma, twenty-five of which occur in child-

ren under fourteen years of age. The incidence of asthma in Canada is approximately 5.8 per 1000 population.

PATHOLOGY

In the early stage, there is spasm of the bronchiolar musculature and edema of the mucous membrane, leaving it pale and boggy. These two processes reduce the size of the lumen, thus hindering the removal of secretions of the mucus, which become tenacious and further fill the tubes. If there is a respiratory infection (which often has precipitated the attack), there is an increase in exudate and vascular congestion which leads to further respiratory embarassment. If the attack continues for a few hours, there may also be atelectasis due to obstruction by tenacious mucus.

CLINICAL MANIFESTATIONS

The duration of the attack before admission to hospital is very variable, but is usually not less than 12 hours and some patients are bedridden for many weeks with continuous bronchospasm before being admitted. In this discussion, an acute severe attack and chronic intractable asthma will both be considered as status asthmaticus. The diagnosis is made by having an asthmatic patient who will not respond readily to treatment. Commonly, nasal congestion with sneezing and watery discharge precede the attack by a few hours or days and the attack begins at night.

On examination, one sees an anxious patient, wheezing and sweating in an orthopneic position, often slightly dehydrated, with a distended chest and a fixed abdomen. Asthma, uncomplicated by right heart failure or pulmonary artery disease, usually does not produce marked cyanosis, even in the presence of severe hypoxia. In a severe attack, the patient may become comatose and die. The respirations are chiefly thoracic with marked use of accessory muscles of respiration. The chest is hyperresonant to percussion and the diaphagm is low as a result of the generalized compensatory emphysema. Throughout the chest, one hears a prolonged expiratory phase and sibilant, sonorous rales which obscure the breath sounds. On examining the chest, one should always be on the lookout for atelectasis which may be indicated by a decrease in the breath sounds and dullness in one area. A sudden rise in the temperature to 101° — 103°F. which subsides within a few hours suggests a bronchus has been blocked by a mucus plug.

Laboratory investigations usually are not much use except in aiding the therapy. However, chest X-ray should be taken, especially in children, to rule out any other condition. The sputum expelled during an attack is characteristically thick and stringy containing small greyish "pearls". When it is spread upon a slide, one sees Curschmann spirals just visible to the unaided eye. In stained smears, there are many eosinophils present (unless the sputum in purulent) along with elongated doubly pyramidal Charcot-Leyden crystals. The sputum should be cultured for pathogenic organisms and antibiotic sensitivity tests should be performed. If the sputum is blood-tinged, one should note if there is any cardiomegaly or inspiratory rales suggestive of cardiac asthma. There may be a moderate increase in eosinophils in the blood, a fact which suggests an allergic basis for the asthma rather than an infective one. The E.S.R. is normal, unless there is a superimposed infection.

TREATMENT

In some cases of status asthmaticus, the simple removal of the patient to hospital will in itself allow the attack to subside. When this happens, it suggests that there are either psychogenic factors which precipitate the attack or many allergenic factors in the patient's environment which impede the effectiveness of antispasmodics. Therefore, the patient should be admitted to an atmosphere from which all inhaled allergens are excluded.

Drugs

The most pressing need is to relieve the anoxia. This can be partially relieved by administrating oxygen which, in itself, contributes to relief of respiratory obstruction.

Bronchodilators and Anti-spasmodics: Epinephrine, ephedrine, and related compounds are the most widely used drugs for attacks of asthma and in most cases are quite effective, especially if given early. Bronchodilators, such as epinephrine, probably act as much by lessening the edema as by relieving the bronchospasm. D. R. Humphreys states that "there is a good deal of synonymity between epinephrine resistant asthma and status asthmaticus, and it is where epinephrine has failed that admission is most often sought". However, true epinephrine-fastness is not common. It is much more common to find a great deal of obstruction by mucus and mucous plugs and when they are cleared, the individual will respond to epinphrine. Therefore, slow subcutaneous injection of a larger dose of epinephrine (up to 2 ml.) over the course of 15-20 minutes is effective when the usual small dose has failed, but where epinephrine resistance has occurred, it is best avoided. Ephedrine probably acts by slowing the rate of destruction of epinephrine and therefore it is not usually successful in status asthmaticus where epine-

phrine resistance has occurred. (In older men in whom there may be some degree of prostatic obstruction, acute retention of urine is precipitated by ephedrine).

Isoprenaline in 1% solution is probably the most effective of the inhaled antispasmodics. The effectiveness of inhaled solutions depends on the size of the droplets, only the smallest below 10 mu. in radius, reaching the bronchioles. Larger droplets are deposited on the mucosa of the respiratory passages whence they must be absorbed and circulated before being effective. Because aerosol medications cause drying and irritation of the tracheal membrane when used too frequently, this form of treatment should be restricted to 4-6 times in 24 hours.

After epinephrine and related compounds have been tried, aminophylline should be the drug of choice. Orally, the use of aminophylline is limited by its slow absorption and by gastric irritation. It should be given intravenously, 0.25-0.50 gms. in 20 ml. saline, slowly (about 1 ml. per minute). If given too fast, syncope and peripheral vascular collapse may occur. In a protracted attack, when response to a single dose is not long maintained, continuous infusion can be employed with a solution of 0.5 gm. in a litre of 5% dextrose given intravenously up to 3 litres a day. Aminophylline may also be given intramuscularly when a vein cannot be found although this is often painful. A 0.5 gm. rectal suppository is another alternative.

If there is no response in 6-12 hours to bronchodilators, hydration, and expectorants, hormonal therapy is indicated. Twenty-five units ACTH per 500 ml. of 5% dextrose intravenously may be given over the course of 6-8 hours once a day for three days. ACTH gel is less effective. Brockbank *et al* suggest the following plan of steroid therapy. Three hundred mg. cortisone acetate, i.e. twelve tablets of 25 mg., are given on the first day followed by a dose decreasing by one tablet each day so that the total course lasts for twelve days. 25 mg. cortisone, 20 mg. hydrocortisone, 5 mg. prednisone or prednisolone, 4 mg. Medrol or Triamcinolone, and 0.75 mg. Dexamethasone are all of relatively equal strength and effectiveness, but the first two are not as useful because they cause sodium retention. This may not be of importance if the steroid treatment is for a short duration but will be of importance if there is any tendency to congestive failure. With this short course the side effects of hormonal treatment do not occur. Dangers, however, are always present, especially in patients who already have a respiratory infection. Therefore, when steroids are used in asthma complicated by a respiratory infection, antibiotics are indicated. A tetracycline may be initiated and altered, if necessary, when bacteriological examination of the sputum has been done. Because of the high allergic tendency in asthmatic patients, penicillin is best avoided. Recently Nystatin 500,000 units t.i.d., p.o. has been given in addition, as monilia appears quite frequently as a cause of oropharyngeal infection.

Sedation: Since the value of sedation is doubtful, one should resist the temptation to sedate the patient who is refractory to treatment. Sedation may either cause mental confusion or, if heavy enough, respiratory and cough suppression at a time when ventilation is impaired. However, when it is necessary to secure rest for the patient, one to two doses of Demerol 50 mg. I.M. may achieve this end. However, prolonged use of Demerol produces cough suppression and drying of bronchial secretions thereby impeding expectoration of mucous plugs. Morphine should never be used because it is a strong respiratory depressant and may lead to respiratory acidosis. The sedative effects of antihistamines are of value, but have no direct effect on asthma. Chlorpromazine 100 mg. I.M. q.6h. is a useful sedative in an acute attack. Measurements made of respiratory function after 25 and 50 mg. chlorpromazine I.M. led to the conclusion that it would induce relaxation quickly with no respiratory depression, and that it was probably the safest sedative to use in status asthmaticus. Occasionally, general anesthetic agents, such as rectal instillation of 30-90 cc. (1-3 oz.) of ether in an equal quantity of olive oil, repeated in 12-24 hours if necessary, may be life saving for the patient. When he awakes, he is, commonly, free of the attack.

Other Measures: Coughing itself aggravates bronchitis and bronchospasm and unnecessary coughing may become a habit to such a degree that it may even induce an attack of wheezing. Some degree of cough suppression may be achieved by the patient's own efforts, but respiratory irritants should be avoided and, if cough is very troublesome, steam inhalation is useful.

Hydration should be started early in a severe attack, for asthmatic patients perspire considerably and cease taking food and water when they are dyspneic. Dehydration may be a cause of inspissation of mucus, and rehydration and humidification of the atmosphere is essential. Some patients can be encouraged to drink adequate fluids; others must be given intravenous saline.

Perhaps the best *expectorant* is potassium iodide which stimulates a mucous secretion which tends to loosen bronchial plugs. It may be given

in doses of 5-15 drops p.o., t.i.d., but it takes 24-48 hours for effect. Before administrating KI, make sure the patient is not allergic to iodides. Syrup of ipecac is useful in children, but if vomiting is produced, other expectorants should be used. Unless the patient is adequately hydrated, expectorants will have a negligible effect.

If the patient is severely cyanosed, oxygen therapy is indicated but one must watch for evidence of respiratory depression due to carbon dioxide retention, evidenced by decreased rate and depth of respiration, and coma. In children, oxygen is best given by tent as use of masks or any other direct means may tend to dry the bronchial secretions, and may be most disturbing to the patient thus producing more apprehension and physical activity. In adults, the oxygen may be given by nasal catheter intermittently, especially if there is a superimposed emphysema present.

Digitalization is indicated in patients who have superimposed cardiac failure. Dyspnea is, of course, accentuated by any degree of circulatory congestion. Cooke recommends avoiding abdominal distension in status asthmaticus since it also accentuates respiratory distress and therefore he suggests giving complete catharsis to these patients. Having the patient in the orthopneic position will also decrease respiratory embarassment.

If the patient's condition deteriorates and his expectoration is gradually becoming scanty or if the sputum contains bronchial casts and each coughing spell results in more wheezing, he may be helped by bronchoscopy and removal of mucus. In asthmatics, this is infrequently performed with I.V. aminophylline administered before, and continuous oxygen during, the procedure; saline lavage may help if mucus is difficult to aspirate. Broom recommends 0.5 gm. thiopentone I.V. and 100 gm. succinyl choline I.V. as the pre-bronchoscopy anesthesia along with intermittent positive pressure breathing. If the tube becomes blocked with mucus, 50 mg. of the calcium salt of trypsin in 10 ml. physiological saline flushed down the endotracheal tube will often clear the mucus. He suggests positive pressure breathing because as the asthmatic respiratory obstruction increases, the therapeutic problem becomes one of ventilation in general, rather than expiration in particular. In the moribund patient, the potential hazards of assisted breathing are less immediate than the actual risks of anoxia. Although "airway trapping" and "airway collapse" may occur with mechanically assisted breathing and may even lead to respiratory collapse, neither is inevitable.

CRITERIA FOR IMPROVEMENT

The patient should have subjective relief as evidenced by a diminishing respiratory effort and cough, and alleviation of the profuse sweating. The chest will no longer, be hyperresonant, expiratory phase should return to normal, and fewer sibilant rales, will be heard. However, if the patient has a superimposed emphysema, the last three signs will still be present. Sputum should disappear in a few days unless the patient has bronchitis.

ACKNOWLEDGMENT — I would like to thank Dr. P.A. Ryan of St. Michael's Hospital for his assistance in the preparation of this paper.

RESPIRATORY DISTRESS IN INFANTS

JOHN D. GOSSAGE, 6T1

Respiratory distress is the most common serious emergency in infants, especially those born prematurely. Two thirds of all infant deaths occur in the first week of life, and of these, more than one half are due to faulty pulmonary ventilation. The early neonatal mortality in Canada is about 9000, and hence the annual loss due to respiratory distress is approximately 3000.

The cardiopulmonary changes which occur at birth are profound, and when they are considered, it is apparent that a failure to adapt to extrauterine existence can result in the Respiratory Distress Syndrome. Prior to birth the lungs are collapsed, and contain amniotic fluid, the pulmonary circuit being low flow/high resistance. At birth, the lungs must expand with air at the first breath, and the pulmonary circuit becomes high flow/low resistance. This alteration is basic to normal respiratory function, and if it does not occur, respiration fails.

Many congenital conditions (diaphragmatic hernia, tracheo-esophageal fistula, etc.) may present as this emergency, as may the respiratory infections.

ANTICIPATION

To anticipate this emergency is to be better prepared to deal with it. The following are the most important situations with which respiratory distress is associated:

(1) Prematurity—the commonest single cause.

(2) Where there has been maternal hemorrhage, sepsis, or toxemia.
(3) Difficult delivery, or Caesarian section.
(4) Infant of a diabetic mother—often premature and delivered by Caesarian section.
(5) Induced labour.
(6) Excessive or wrong sedation or anesthetic to the mother—a particularly unfortunate, and all too common cause.

It is obvious that many of the above are preventable by proper maternal and infant management.

SIGNS OF RESPIRATORY DISTRESS

Careful examination of the infant at birth is important, and is, unfortunately, seldom done. Such examination puts us in a much better position to deal promptly and efficiently with any emergency, and particularly respiratory distress. The Apgar rating is very desirable in that it attempts to assess cardiopulmonary and neurological vectors, both important in the evaluation of respiratory function. (fig. 1) Infants with low Apgar ratings notoriously get into trouble more frequently than those with high ratings.

The important signs of respiratory distress in infants are laboured breathing—the respiratory

	0	1	2
Heart Rate	Absent	under 100	over 100
Resp. Effort	Absent	Weak Cry, hypo-ventilation.	Good, strong cry.
Muscle Tone	Limp	Some flexion of extremities.	Well flexed.
Reflex Irritability	None	Some motion	Cry
Colour	Blue, pale.	Body pink, Extremities blue.	Completely pink.

Rating to be taken within 60 seconds of birth.
Total possible— 10

FIG. 1 — THE APGAR RATING

	HIGH OBSTR.	LOW OBSTR.
Resp. distress	Inspiratory	Expiratory, inspiratory
Stridor	Present	Usually absent
Hoarseness	Present	Usually absent
Chest	Underaerated	Overaerated
Diaphragm	High with maximal excursion	Low with minimal excursion
Resp. Rate	Slightly increased	Markedly increased
Retraction	With Inspiration	With inspiration, but less
Laryngeal Obstr.	Present	Absent

FIG. 2 — DIFFERENTIAL DIAGNOSIS OF HIGH AND LOW OBSTRUCTION

rate initially being rapid at 60/minute or more, and later slowing, with periods of apnea of increasing frequency and duration — cyanosis, flaring of alae nasi, intercostal and subcostal retraction, edema — often associated with pulmonary edema. It is necessary to distinguish carefully between high and low obstruction, as the two are not treated in the same manner (fig. 2).

The only object of the emergency treatment is to get oxygenation of the tissues re-established. X-rays, ECG's, and other aids to diagnosis do not help in the immediate saving of a dying baby's life, and should not be used at this critical moment unless the results they give will directly affect the emergency management.

EMERGENCY TREATMENT

The object of emergency treatment is the re-establishment of as nearly physiological respiration as can be achieved in the shortest possible time. This requires an airway, an adequate oxygen concentration in the atmosphere supplied to the child, and minimal hindrance to gaseous exchange at the alveolar level.

An *airway* is the first requirement for normal respiration. It may be obstructed at any level, the obstruction may be fluid or solid, and due to foreign body, inflammation, or external compression of the trachea.

Airway obstruction is overcome either by removal of the material causing it, as with foreign bodies and newborns obstructed with mucus, or by-passing it. Fluid obstruction is handled by inverting the baby, and assisting drainage by the use of suction, either oral or mechanical. Postural drainage is an important principle in removing fluid from an airway. Solid obstruction is a more difficult problem to handle, unless it is a foreign body lodged within reach of forceps. If elevation of the chin fails to restore normal respiration, an airway must be inserted. In the majority of cases of high obstruction, this is sufficient to overcome the obstruction. Tracheotomy is an exceedingly difficult procedure with infants, and should not be done except by someone experienced in doing it. Such persons are generally only available in children's hospitals. However in the dire emergency, where all else has failed, it may be all that is left which can overcome high obstruction.

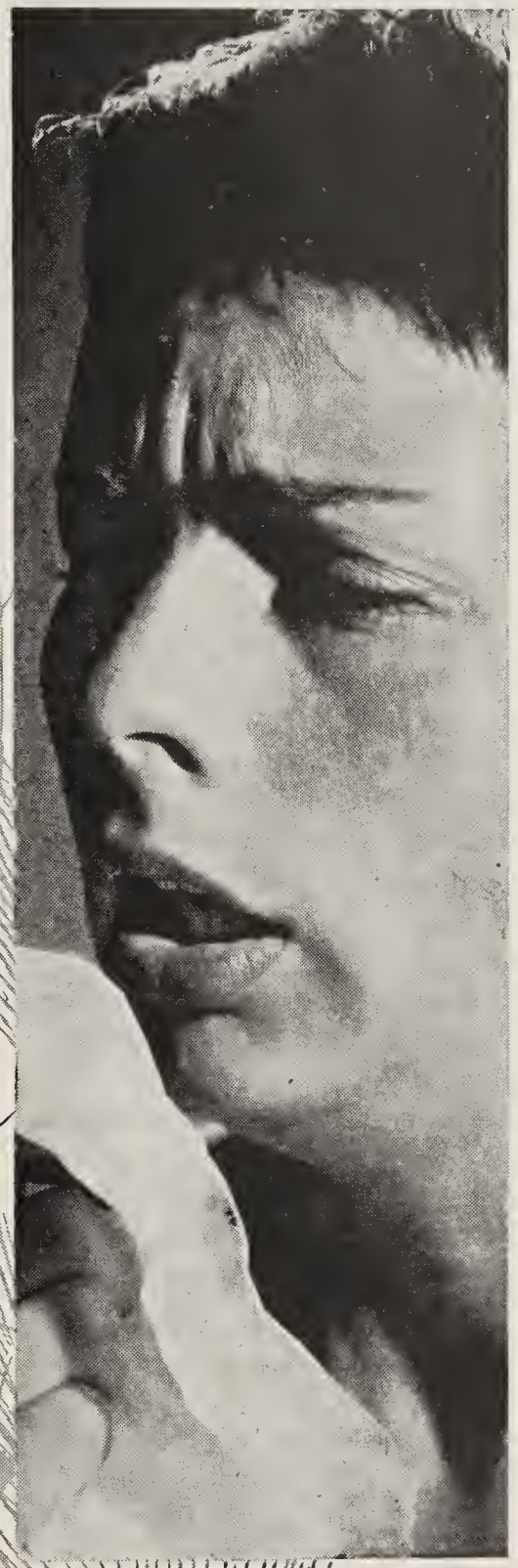
The Oxygen Supply: Most infants in respiratory distress require an oxygen-enriched atmosphere for relief. Cool moist oxygen, as supplied in the Isolette and similar pieces of apparatus, has been found the most satisfactory. Not only does it administer oxygen, but the moisture in the atmosphere helps to loosen sticky, adherent secretions from the lower bronchial tree which are present in many cases of low respiratory obstruction, such as bronchiolitis, pulmonary edema, and the pneumonias. Even in very small babies such as prematures, very high concentrations of oxygen may have to be used if the child's life is to be saved. For many years, concentrations of oxygen above 40% have not been used with small babies because of the danger of retrolental fibroplasia. However recent work suggests that the occurrence of this condition is a function of both the O2 concentration and the length of time during which it is administered. Hence, high concentrations can be safely given over shorter periods of time, and such concentrations may be required to save the infant. The oxygen must be able to get to the alveoli to be of any use. Therefore, if the baby is unable to breathe adequately on its own, assisted respiration may have to be introduced, as with manual artificial respiration or the Bird respirator. The place where such emergencies are handled should have a supply of pure oxygen available.

The Alveolar Level: For gaseous exchange to occur, the alveoli must be able to expand with air. Conditions such as pneumothorax and pyopneumothorax are particularly dangerous in the infant because of the much greater mobility of the mediastinum, which shifts readily to the opposite side, thereby impeding air entry to both sides of the chest. Fortunately, the history and physical signs of such catastrophies are characteristic. In such cases, re-expansion of the affected side by introduction of a needle is justified, and should be carried out as asepically as possible. In most cases, obstruction of exchange at the alveolar level is due to material within the alveoli, as with the pneumonias, and, again, physical examination will elicit this as the cause. In such cases, general measures to limit the infant's need for oxygen must be instituted. The management of the acute asthmatic attack, where inflammation at the bronchiolar level causes a ball valve mechanism to inhibit gaseous exchange, is covered elsewhere in this issue.

General measures which can be commenced in the emergency situation are as follows: (1) Correction of fluid loss and/or electrolyte imbalance. (2) Where fever — anti-pyretics and cool sponge baths. These make the infant more comfortable, and reduce his O2 requirement. In infants, *sedatives* are, except in rare circumstances, contraindicated because of the immaturity of the detoxifying organs in this age group.

SUMMARY

Respiratory distress is a common pediatric emergency. Certain conditions predispose to it occurring, and one should be on the lookout for it in these cases. The signs of respiratory distress are enumerated, and the differential



COUGH
responds to treatment with "PINOCODEINE"(N) BRAND

Each fluid ounce contains:

Pinus strobus.....	32 gr.	(2.1 G.)
Prunus virginiana.....	32 gr.	(2.1 G.)
Sanguinaria canadensis..	4 gr.	(0.25 G.)
Populus balsamifera.....	2 gr.	(0.12 G.)
Chloroformum.....	1 min.	(0.06 cc.)
Codeinae phosphas	1 gr.	(60 mg.)
Flavoured syrup base.....		q.s.

(N) Telephone narcotic prescription permitted.

DOSAGE: One or two teaspoonfuls every four hours.

Packaged in 16 fluid ounce bottles.

Charles E. Frosst & Co.
MONTREAL CANADA

Frosst

diagnosis of high and low obstruction is discussed. The principles of management are provision of an airway, and oxygen supply, and facilitation of alveolar exchange by specific and general means. Finally, there are certain general measures which can be commenced immediately which aid the emergency management considerably.

ACKNOWLEDGMENT — The interest and help of Dr. H. Bain, and the members of the respiratory team at the Hospital for Sick Children are gratefully acknowledged.

THE ACUTELY DEHYDRATED INFANT

P.W. TRUSCOTT, 6T2.

The most important causes of dehydration in infants are diarrhea and vomiting. Diabetic acidosis is also a major cause. These three states involve a severe loss of water and electrolytes. An inadequate water intake may also give rise to dehydration if it is below the amounts needed to compensate for the obligatory water of the kidneys, evaporation at the lungs and insensible perspiration. Water only may be lost in excess from the lung or skin in states of hyperpnea and fever.

Diarrhea: The mild states of diarrhea present no problem to the physician because the dehydration is usually corrected with oral fluid therapy in the home. The problem is deciding when a case is severe enough to require intravenous fluids in the hospital. The physical state of the child should be sufficient to indicate when the physician should use intravenous therapy. The signs and symptoms of mild diarrhea contrasted to those of a severe case of diarrhea follow:

MILD DIARRHEA

1. Appears unwell.
2. Sight fever.
3. Up to ten loose water stools daily.
4. Skin is pink and warm.
5. The child is thirsty.
6. May have decreased urinary output.
7. Pulse rate is rapid.
8. The child cries because of abdominal cramps.
9. May retain part of oral fluids.

SEVERE DIARRHEA

1. Obviously ill with or without clinical dehydration.
2. Rectal temperature may be very high.
3. Very frequent large explosive stools.
4. Skin is pale and cool. Lips are cyanosed.
5. Thirst may be marked or absent.
6. Oliguria is marked.
7. Pulse thin and rapid at 160-180 per minute.
8. The infant is restless and whines.
9. Refuses fluids and vomits them quickly.
10. Respirations are deep and sighing but rapid.
11. Obvious signs of dehydration appear including: depressed fontanelles, sunken eyes, parched mouth, skin loose and inelastic.
12. If it progresses untreated, the patient may go into a coma and ahow signs of circulatory collapse.

Three major considerations must be assessed by the physician treating severe diarrhea: First, how much fluid and electrolytes have been lost? Second, how much fluid and electrolytes does the child need to supply his daily requirements? Third, how much excess water and electrolytes will the child need to offset the loss due to continuing disease?

The answer to the first question is found in the history, but the physical examination gives valuable additional information. As a rule it will not be necessary to await laboratory data to decide on the amounts of fluids and electrolytes that will be administered. The brief history should include a description of the quantity and kind of fluid taken in and lost. The physical examination will reveal most of the points listed above under severe diarrhea. A good rule of thumb for quick and accurate assessment of fluid loss in the infant is to subtract the weight at examination from the weight of the child just previous to the disease. This figure in grams equals the fluid lost in cubic centimeters. Only 50% of the volume lost by diarrhea should be replaced in the first twenty-four hours. In answer to the second question, the normal daily requirement of an infant is 60-75 c.c. per lb. per day. As question three is not strictly within the realm of emergency measures, it suffices to say that careful estimates of losses in continuing diarrhea at the hospital will provide the answer.

Assuming that the physician first sees the infant at home, he must know how to deal with the child in that situation. Having made the diag-

nosis of severe diarrhea, the infant should be removed to hospital immediately. If it is physically impossible to do this and the child is in a collapsed state he should be given 2/3 5% glucose in water and 1/3 N. saline by syringe (10 c.c./lb.) over a half hour period. It is advisable for physicians practising in remote areas to have the necessary equipment readily available.

Once in hospital an intravenous infusion is started immediately by stab or cut down if necessary. Having estimated the amount of fluid required, a solution consisting of 2/3 5% glucose in water and 1/3 N. saline is administered. The above should be given by I.V. drip at the rate of 30 c.c./lb. in the first 3-6 hours and the remaining fluid should be infused uniformly during the balance of the twenty-four hour period.

The above measures will be modified by the addition of potassium in the infusion if diarrhea has been present for three days or more. It will also be modified by the administration of sodium lactate if the child is acidotic with signs of hyperpnea. However, the above two measures should only be carried out after adequate urinary output is established. Supportive therapy must include oxygen and warmth from the outset.

Vomiting: In certain conditions vomiting may accompany diarrhea. Usually the loss of base in the diarrhea is far greater than the loss of chloride in the vomitus. In this case a metabolic acidosis and dehydration develop as in diarrhea *per se* and thus treatment is the same as that outlined under diarrhea. However, if vomiting should be the predominant factor, or if it should occur alone, the greater loss of fluids and electrolytes will occur from the stomach. Another common cause of gastric vomiting in the infant is pyloric stenosis. The electrolyte loss involves mainly chlorides. This leads to a metabolic alkalosis. A severe case of metabolic alkalosis may be accompanied by an excessive loss of potassium. Also, hypopnea is marked in this state.

Treatment for this condition consists of administration of 2/3 5% glucose in water with 1/3 normal saline. One half of the estimated fluid deficit is given in the first twenty-four hours. Added to this, one administers the same solution for maintenance at a rate of 50-60 c.c. per lb. body weight per day. Potassium chloride can be added to each bottle of electrolyte solution if the urinary output is good. The amount used in 0.1 G. per lb. body weight per day.

If the metabolic alkalosis is very severe and accompanied by tetany calcium gluconate is given as often as is required in amounts of 5-10 c.c. of a 10% solution in 40 c.c. normal saline (I.V.) over a 10 minute period.

Diabetic Acidosis: Diabetic acidosis and coma are medical emergencies requiring immediate treatment. The signs and symptoms of diabetic acidosis are nausea, vomiting, hyperpnea, acetone odor on breath, a dehydrated appearance and drowsiness progressing to unconsciousness and coma.

For the child under one year 5 units of regular insulin should be administered subcutaneously *stat.* If the patient is unconscious, an additional 5 units of regular insulin should be given I.V. *stat.* Intravenous fluid therapy should consist of one part M/6 sodium lactate and two parts isotonic sodium chloride administered at the rate of 500-1000 c.c. in 1-2 hours to relieve the dehydration. In a case of severe acidosis in which the CO2C.P. is below 8 mEq./L., sodium lactate is best given as follows: 2 c.c. M/6 lactate per lb. body weight to raise the carbonate level by 1 mEq./L. One calculates the amount required to raise the carbonate level to 18 mEq./L. and the kidney accomplishes the rest of the adjustments.

The stomach is emptied with a tube to prevent aspiration pneumonia. The patient should be kept warm. If the blood pressure remains at a low level blood or plasma may be required to overcome shock. Appropriate antibiotics should be administered if infection is present. Further treatment involves following the blood glucose levels every 2 hours and administering insulin according to the blood sugar levels. Urine samples should be followed hourly and tested for glucose and acetone. When the blood sugar falls to 200 mg.%, I.V. therapy should be altered to 2/3 5% glucose in water and 1/3 N. saline.

Infection: Infection is usually accompanied by fever which gives rise to an increased loss of fluids through perspiration and hyperpnea. Added to this there is usually a decreased intake of fluid as a result of malaise. The combination of the above two factors results in a hypertonic dehydration. It is difficult to decide whether or not solutions of low sodium or high sodium content are best to use as convulsions have been reported with both. The solution of 2/3 5% glucose in water and 1/3 normal saline will prove to be the best in most cases. The total requirement for one day estimated as shown for diarrhea is given in the first eight hours. The remainder should be infused uniformly throughout the balance of the day.

One must be fully aware that the problem of dehydration in infancy is quite different from that in the adult. Water represents 70-75% of the body weight in the infant whereas in the adult it is only 60% of the total weight. In a 15 lb. infant,

40% of his extracellular fluid is exchanged each day while a 154 lb. adult only has 14% of his E.C.F. exchanged. Thus dehydration occurs much more easily in the infant than in the adult.

The secondary disturbances in water and electrolyte balance result in an emergency and a real threat to the life of the patient, while the underlying disease may be relatively unimportant in this respect. Only a thorough understanding of these secondary changes can insure a successful outcome in the treatment of the acutely dehydrated infant.

ACKNOWLEDGEMENT — The author wishes to express his appreciation to Dr. M. Gerstein for his suggestions and assistance in the preparation of this paper.

ECLAMPTOGENIC TOXEMIA

D.J. Mc DONALD, 6T1.

Pre-eclampsia and eclampsia are clinical divisions of a disease peculiar to gravid and puerperal women. This condition is recognized to be an acute vascular disease manifesting generalized arteriolar spasm, and characterized clinically by edema, hypertension and proteinuria. Eclampsia is characterized, in addition, by the appearance of tonic and clonic convulsions, followed by more or less prolonged coma. Further deterioration of this condition frequently results in death.

PATHOGENESIS

The basic physiochemical disturbances are:

1) Sodium and water retention, and
2) Arteriolar spasm.

Theories of Etiology

1) Vasopressor substance liberated from uterus, decidua or placenta,
2) Endocrine imbalance.

DIFFERENTIAL DIAGNOSIS

The typical eclampsia of pregnancy is not difficult to diagnose. The presence of marked edema, proteinuria and hypertension occurring in a pregnant woman in the last trimester, associated with convulsions and coma, presents a rather clear clinical picture. One must be more cautious with a patient whose past history is unknown, with little or no edema, and whose blood pressure is not elevated. Idiopathic epilepsy, diabetic coma, cerebrovascular accidents, uremia, trauma and poisoning must be carefully considered and ruled out.

TREATMENT OF ECLAMPSIA

Preventive

The warning signs of abnormal gain in weight, edema, hypertension and albuminuria are easily detected. Frequent prenatal visits are essential and treatment must be instituted early.

Hospitalize the patient immediately if:

1) Blood pressure greater than 150/100 or rise in systolic over 30 mm. Hg., or rise in diastolic over 15 mm. Hg.
2) Proteinuria
3) Sudden edema

If a favourable response occurs, carry the patient to the thirty-fourth week and then terminate pregnancy by rupturing membranes, pitocin drip or Caesarin, depending on the state of the cervix, pelvis or presentation.

If an unfavourable response to treatment occurs, as indicated by:

1) proteinuria greater than 5 gms/litre
2) oliguria
3) increasing blood pressure
4) sudden edema
5) increasing NPN
6) severe headache
7) visual disturbances
8) sudden onset of vomiting and severe epigastric pain in this case, terminate the pregnancy.

Treatment of the Convulsive State

1) Admit to a dark and quiet room
   (a) Place a blood pressure cuff on one arm — BP every 15 to 30 minutes
   (b) Indwelling Foley catheter — to determine hourly output
2) Preparation to prevent injury
   (a) Mouth gag
   (b) Side boards on bed
3) House surgeon assigned to patient on admission, and he assumes responsibility for continuous supervision.
4) Aspiration apparatus available for removal of mucus from throat.
5) Oxygen administration by nasal catheter or face mask or by intubation if necessary. Tracheotomy to be performed in necessary.
6) Sedation:—

   Start — I.V. Sodium Amytal 7 1/2 grains

Withdraw blood for CO2CP, NPN, uric acid, ESR S.C. Pantopon — 1/3 grain
1 hour — Repeat Pantopon — 1/3 grain S.C. Sodium Luminal — 2 grains I.M.
3 hours— Pantopon
7 hours— Sodium Luminal
13 hours— Sodium Luminal
21 hours— Sodium Luminal
—or—
Mg. SO4 — I.M., 10 cc. of a 50% solution in each buttock for an initial dose of 10 Gm., followed by 5 Gm. every 8 hours. Phenobarbital 1.5 grains q8h. — Alternate with Mg. SO4 so one is given every 4 hours.

7) Vasodilator (Antitensive) Therapy
Crypenamine (Unitensin) and 1-hydrazinophthalazine (Apresoline) may be given I.V. singly or in combination. Check B.P. and pulse rates every 5 minutes for first hour and then every 15 minutes. Regulate the vasodilator infusion to maintain the blood pressure at approximately 140/90.
8) If Urinary output is less than 500 cc. in 24 hours, I.V. Aminophylline is to be considered.
9) If CO2CP is less than 20%, I.V. NaHCO3, 5% buffer is indicated.
10) When eclampsia is under control, and if the patient is not in labour, membranes are to be ruptured in bed, with anesthesia, if necessary.
11) If rupture of the membranes is contraindicated due to:
(a) Condition of cervix,
(b) Age of patient,
(c) Primigravida, or
(d) Obvious disproportion due to deformity, — Caesarian section is to be performed.
12) The second stage of labor in any case should be short. Forceps should be used if indicated.

Treatment of underlying Disease

1) Diuresis
(a) I.V. fluids — A minumum of 1000 cc. of 5% glucose in water are given every 8 hours, for a total of 3000-4000 cc. every 24 hours.
(b) Vasodilator
(c) Termination of pregnancy

VAGINAL HEMORRHAGE IN LATE PREGNANCY

L.M. JERRY, 6T1.

Vaginal hemorrhage in the second half of pregnancy is a common symptom; 1 in 20 pregnant patients experience such hemorrhage from a pathological cause. This paper will attempt to present in a practical fashion the diagnosis, investigation and management of such hemorrhage with particular reference to the three commonest gestational states implicated in its causation; namely, placenta previa, abruptio placentae, and rupture of a placental marginal sinus.

PROPHYLAXIS

"An ounce of prevention is worth a pound of cure". Direct your attention to the following:

1. A gravida who suffers from other systemic disease will be worse off should she have a hemorrhage than will a healthy gravida. Hemorrhages tend to recur in succeeding pregnancies.
2. Some authorities delay extensive internal pelvimetry until the seventh or eighth month, especially with a history of recent bleeding or of a previous abortion, in order to avoid a possible abortion induced by the manipulation. Relaxation of the pelvic structures in the later months will afford the obstetrician more precise information at a time when the patient is better adjusted psychologically to his care.
3. The anemic patient cannot withstand blood loss well if her hemoglobin is already below 13 gm.%. Repeat the hemoglobin estimation at the 24th and 36th weeks.
4. Determine the patient's blood group (ABO and Rh). If you think that the patient may require a Caesarian section, have blood on hand when she is admitted in anticipation of a possible emergency.
5. Tell your patient to contact you immedicately if during her pregnancy she experiences any vaginal bleeding at any time in any amount. Remember, serious bleeding can be painless.

INITIAL MANAGEMENT

When your patient telephones to tell you about her bleeding, do not waste time in paying her a house call; rather, advise her to come to hospital immediately where you will see her as soon as she arrives and admit her to a single unit on the labour floor.

Since you must estimate the blood loss largely from the patient's story, you may be grossly in error. Are uterine contractions present? Is the uterus tender? Is it relaxed? Record the mother's temperature, pulse rate and blood pressure as well as the rate and quality of the fetal heart. Abdominal examination should be gentle.

Obtain a sample of venous blood for routine hemogram, cross and typing, clotting time, qualitative fibrinogen test and, if necessary, quantitative fibrinrogen determination.

Pelvic and rectal esaminations are contra-indicated. You may precipitate hemorrhage from trauma to a low-lying placenta, or stimulate the onset of uterine contractions before the patient has reached term.

The patient may not have a shower or bath. A perineal shave is permitted if bleeding is minimal.

Record at frequent intervals:

a) maternal blood loss
b) maternal temperature, pulse rate, blood pressure.
c) Consistency of the uterine fundus, its relaxation and its tenderness.
d) the presence and character of uterine contractions.
e) the rate and quality of the fetal heart.

Give the patient nothing by mouth in case anesthesia is required. Have a trained anesthetist to administer the anesthetic, but avoid spinal anesthesia which may induce a fall in blood pressure. A properly equipped recovery room should be available for the patient after delivery.

Remember that only a ruptured uterus or fetal distress in the presence of severe abruption require emergency surgery. Since these are rare events, most cases of hemorrhage do not require the use of a delivery or operating room for immediate surgery or diagnostic procedures. The problem, rather, is to effectively treat the shock incurred by blood loss by intravenous replacement of whole blood. Prompt diagnosis of the cause is of secondary importance relative to replacement of blood volume.

DIAGNOSIS AND INVESTIGATION

Any woman presenting with vaginal hemorrhage in the late months of pregnancy has placenta previa, abruptio placentae, or rupture of a placental marginal sinus until proven otherwise. Most do not have a serious lesion. A differential diagnosis of such a hemorrhage is shown in Table 1.

PLACENTA PREVIA

History: The bleeding occurs suddenly without warning in the second half of pregnancy, recurring often, in a health gravida who may awake in bed in a pool of blood. It is usually bright red and painless, and may be spotty or profuse. The hemorrhage is rarely fatal unless aggravated by a rectal or vaginal examination.

Examination: The uterus is soft but not tender. If labor has begun, the resting tone of the myometrium is normal. In nearly 1/3 of cases the low-lying placenta induces either abnormal fetal presentation (breech, oblique and transverse lies) or a presenting part which lies unexpectedly high and free above the pelvic brim.

Sterile Vaginal Examination: This is the most definitive of the special investigations.

Indications:

Employ when clinical signs and symptoms point to the desirability of immediate delivery.

i) profuse hemorrhage at any stage of pregnancy.
ii) onset of labor.
iii) Significant bleeding in patients at or near term.
iv) Fetus of viable size.

A gentle intracervical digital examination is performed to find the placental edge and to determine its relationship to the internal os as well as to assess the general condition of the

Table I: Differential Diagnosis of Vaginal Hemorrhage in Late Pregnancy

Extragenital: hematuria, anorectal bleeding.

Genital:

Associated with pregnancy	Not associated with pregnancy
show	cervicitis and erosion
placenta prévia	general trauma and lacerations
abruptio placentae	foreign bodies
rupture of a marginal sinus	genital infections
varicosities	tumors — polyps
chorionepithelioma	— carcinoma
hydatidiform mole	blood dyscrasia

cervix, its dilatation and its effacement. The placenta is characteristically spongy and gritty to the touch. Palpation of the lower segment through all the vaginal fornices demonstrates the presence of a soft mass lying between the examining finger and the presenting part. The presenting part may ride high or be deflected laterally, anteriorly or posteriorly. Moderate suprafundic pressure will not cause a significant descent of the presenting part or reorientation of its axis.

This examination renews or exaggerates the bleeding which may become torrential. Have plenty of cross-matched blood immediately available. Have an intravenous running in at least one vein through a large bore needle. Do the examination in an operating room under double setup with preparations for Caesarian section or vaginal delivery with the surgical team and anesthetist ready. If placenta previa is strongly suspected give the anaesthetic first.

If the above indications which are urgent enough to prompt an immediate sterile vaginal examination are lacking, precede such an examination with one or more of the following investigations.

Radiologic Diagnosis: Theoretically a routine investigation requires recumbent A.P., erect lateral, and recumbent soft tissue lateral films with a semi-reclining lateral if indicated. This is a lot of radiation for a fetus, so that the patient is first "screened" by taking a single standing soft-tissue lateral film. Contrast radiography may be used employing a lateral film in conjunction with a cystogram or air in the rectum for anterior or posterior implantations respectively. A presumptive diagnosis of placenta previa may be made in 75 — 90% of cases by demonstrating;

i) a placental shadow in the lower uterine segment (direct placentography) Calcific deposits help to localize the placenta.

ii) a deviated or high presenting part under the action of gravity (indirect placentography). A full rectum, bladder or pelvic tumor must be differentiated.

Sterile Speculum Examination: If placentography shows a fundal placenta, insert a speculum, do a Papanicolaou smear, do not do an intracervical digital examination. If no pathology is seen and the bleeding has stopped, send the patient home. If placentography shows a low-lying placenta insert a speculum and gently palpate the fornices only.

If none of the above studies exclude placenta previa conclusively, keep the patient in hospital and give her expectant management for placenta previa.

Bleeding usually stops spontaneously. If the patient is at term and no cephalo-pelvic disproportion or indications for immediate delivery exist, do the sterile vaginal examination (following the other investigations) on the second or third day of admission after blood loss has been restored. If placenta previa is found, proceed with definitive treatment. If the fetus has not yet reached the stage of viability (36 to 37 weeks) and bleeding has stopped, delay the sterile vaginal examination but perform the other investigations. If placenta previa is presumptively found, employ expectant management to bring the patient as close to term as possible.

ABRUPTIO PLACENTAE

History: The closer abruption occurs to the twentieth week, the closer it resembles an abortion clinically. The patient complains of bright red or dark vaginal bleeding which may be spotty or profuse, and which is accompanied by abnormal uterine pain. Some cases are painless; 20% have concealed hemorrhage with no external bleeding. There may be symptoms of toxemia.

Examination: The uterus is tender and either tetanic or poorly relaxed between contractions if labor has begun. Signs of toxemia may be present. Nearly 20% of cases show some coagulation defect which can be best recognized by the obstetrician who views abruption with a high index of suspicion. Look every 30 to 60 minutes for persistent oozing from all points of trauma (especially venapuncture sites, abdominal wound, episiotomy site, gingiva, purpura), and for persistent fluidity of the extravasated blood (observe the blood on the delivery room floor!) Cases may be classified clinically as shown in Table II. Suspect all cases as severe once you diagnose abruption since mild cases may suddenly become severe, and since nearly 40% are already severe (shock, coagulation defect, prolonged tetanic contraction of the uterus, complete placental separation).

Clot Observation Test: To rectify the presence of a coagulation defect place 5 ml. of maternal venous blood in a 15 ml. test-tube. Gently agitate 4 or 5 times or invert, label with the time and the patient's name and attach to the wall above the patient's bed with adhesive tape along with a control from a nurse or intern. The test is abnormal if no clot forms in 6 minutes, or if after 30 minutes the clot breaks up when the tube is shaken, or if the clot is not solid and lyses within one hour. After retraction the clot

should occupy 35 to 45% of the specimen of blood. Repeat every 30 to 60 minutes until delivery and for the first 4 to 5 hours after delivery. Most fibrinogen estimations fall no lower than 150 mg. %. The critical phase of hemorrhage with uncoagulable blood and shock occurs at a fibrinogen level of 90 mg.%. See Table III for further evaluation of the test.
Qualitative Fibrinogen Test: Estimate repeatedly the fibrinogen content of maternal venous blood using Fibrindex every 30 to 60 minutes until results are normal. Report as normal or subnormal (fibrinogen level less than 150 mg.%).
Quantitative Fibrinogen Test: This determination takes too long to be of immediate value in management. It serves to confirm the accuracy of the other tests and of your diagnosis in retrospect.

• • •

All tests are required including repeated observations for hemorrhagic diasthesis since only one test may be abnormal. It is wise to chart observations of the patient's progress on a flow sheet, an example of which may be found in the J.A.M.A.; 170:1520, 1950: Flow Sheet for Use in Obstetric Coagulation Problems; Caillouette, J.C., Longo, L.D., and Russell, K.P.

RUPTURE OF A PLACENTAL MARGINAL SINUS

Usually 1 in 20 patients experience pathological hemorrhage in the third trimester. Roughly 40% of these are due to a ruptured marginal sinus. 30% are due to placenta previa and 10% to abruption. Rupture of a marginal sinus represents nearly 1/3 of all hemorrhages of late pregnancy and 1/2 of all painless hemorrhages of late pregnancy. Most (40%) occur during labor. 35% are concealed (compared to 50% concealed in abruption).

Clinically the condition resembles placenta previa. Typically the patient awakes with a desire to void accompanied by abdominal discomfort. When the woman gets up to micturate, hemorrhage occurs into the toilet. The bleeding is either painless or accompanied by a dull aching or nagging sense of uterine pressure. Only 5% have uterine tenderness or a moderate increase in uterine tone. Shock is rare. 8% of cases occur in conjunction with a circumvallate placenta. Retroplacental trapping of blood in abruption seems to lead to uterine hyperirritability. The same phenomenon in marginal sinus rupture predisposes rather to an increased incidence of premature labor within the first 24 hours following the hemorrhage. The diagnosis is confirmed only at delivery when a fresh clot which is only several hours old is found within the gaping sinus.

MANAGEMENT

Placenta Previa

Expectant Management: Indications are all of:

a) Labor has not ensued.
b) Gestation is less than 36 weeks.
c) Bleeding is minimal or has stopped.

Approximately 25% of cases can be managed expectantly. One is justified, however, in employing such treatment only under ideal circumstances. Follow-up care must be meticulous; the patient must be intelligent and fully co-operative. Adequate facilities for rapid treatment and blood replacement must be available at all times. In other words, ideally, the patient is kept in hospital. This may not be practical, in which case the mother may go home provided that she understands the risk involved and the necessity of getting to hospital quickly should bleeding recur. A multiparous patient may be loath to leave her children alone in the event of an emergency. Consider, as well, how far she must travel, what transportation facilities are available, etc. The digital intracervical examination is postponed until expectant management is terminated, but should always be done just before delivery even if the patient has been asymptomatic for a while. The other special investigations cited above should be done. Vaginal manipulation, douching and intercourse are probhited. Blood volume is restored, and the hemoglobin estimation is followed at frequent intervals.

Active treatment is immediately begun if:

(a) Labour begins
or (b) The fetus reaches maturity (36 weeks or more),
or (c) Persistent or recurrent hemorrhage occurs.

Definitive treatment takes precedence over the use of repeated transfusions.

Active Management

Indications are one or more of:

(a) labour has begun.
(b) The fetus is mature.
(c) hemorrhage continues uncontrolled.
(d) expectant management has failed or terminated.

Control the hemorrhage and replace blood adequately. Cross and type for at least 1500 to 2000 ml. Prepare the O.R., then take time to collect your thoughts. Carry out investigations as outlined above depending upon the urgency of the situation. Prepare to deliver the patient at a time optimum for maximum maternal and perinatal salvage. Choose a mode of delivery that minimizes lacerations to the cervix and lower uterine segment at the vascular, soft, pliable placental site. Precede delivery with the

	Grade O: Subclinical	Grade I: Mildly Severe	Grade II: Moderately Severe	Grade III: Severe
Vaginal Bleeding	o	o or slight (under 400 ml.) No signs concealed hemorrhage.	Slight to moderate (over 400 ml.) May be entirely concealed.	Usually moderate to excessive, Continuous; interval or occult
Myometrium	Normal	Slight increased sensitivity, hyperirritability Not tetanic	Hypersensitive Moderate uterine tetany	Hypersensitive, tetanic, board-like, sensitive to pressure
Maternal Shock	o	o	o May be maternal tachycardia	Severe, out of proportion to blood loss. B.P. variable
Fetal Death	o	o	Yes or No FHR-o or distressed	Yes
Clot Observation Test	Normal	Solid Clot, no lysis	A fair clot forms which undergoes partial lysis after 1 hour.	Positive; No clot or flimsy clot with lysis within 30 minutes.
Pathology	Microscopic lesions	No more than 1/6 of placental surface involved with adherent clots.	Between 1/6 and 1/3 of placental surface involved.	complete or almost complete separation.
Signs and Symptoms	o	Mild abdominal pain, slight tenderness over placental site.	Sensation like something ruptured or gave way, followed by sensational pressure, then uterine pain & tenderness	as in Grade II with shock and coagulation defect.

Table II: CLINICAL CLASSIFICATION OF ABRUPTION

Clot	Lysis	Fibrinogen conc. in mg.% plasma	Clotting Time	Fibrinogen conc. in mg. % plasma.
no clot	o	under 60	6 min.	approx. 150
flimsy clot	complete in 60 min.	60 – 120	6 min. with poor clot	approx. 100 – 150
firmer clot	partial in 2 hrs.	120 – 150		
firm clot	No lysis	over 150	No clot in 30 min.	under 100

Table III: TWO METHODS OF EVALUATING THE CLOT OBSERVATION TEST

digital intracervical examination. Vaginal delivery is indicated in infected cases and the multiparous patient with minimal marginal placenta previa and implantation on the anterior wall with a ripe (dilated 4 cm. or more) cervix in whom hemorrhage is easily controlled. Rupture of the membranes combined with traction on the scalp (Willett's clamp) or on the foot of the infant depending on the presentation will staunch hemorrhage.

Delivery by Caesarian section is indicated, in essence, for all other cases. A classical section may be done to avoid the placental site, or a transperitoneal lower segment section can be done. In the latter, peel the placenta off the uterine wall rather than incising it.

Infection and lower nephron nephrosis are the handmaidens of hemorrhage. Should infection supervene (fever, foul discharge, uterine pain and tenderness, history of vaginal manipulation), transfusion, antibiotics and delivery *per vaginam* are indicated.

Abruptio Placentae

The need for blood is usually greater than is appreciat ed. Intravenous fibrinogen must be given for hypofibrinogenemia. To stop the clotting defect the uterus must be emptied. The severity of the depression of the fibrinogen level is directly proportional to the severity of the clinical symptoms. Natural regeneration of fibrinogen occurs promptly after the uterus is emptied.

Antibiotics should be used in the presence of hemorrhage or uterine apoplexy to prevent infection.

Deliver the patient within 10 to 12 hours. A brief atraumatic labour is optimum. The state of blood coagulability is the most important factor to consider when deciding on the method of delivery. A *couvelaire* uterus is not a mandatory indication for hysterectomy.

No operation (except amniotomy) must be carried out until hypovolemic shock and hypofibrinogenemia are corrected.

Mild cases past 36 weeks gestation, and all moderate and severe cases should be delivered as soon as safety allows, since the severity of the condition varies directly as the time elapsed between the onset of hemorrhage and the delivery. Consider expectant management in mild cases of less than 36 weeks gestation with due regard to the mother's safety in an attempt to obtain a viable baby.

Management of the Severe Case: In the first hour, cross and type for 1500 to 3000 ml. of whole blood and prepare 10 gm. of fibrinogen. The need for blood is usually underestimated since hemorrhage may be concealed. Estimation of blood loss is based on assessment of the patient and her history. Be generous and then double your estimate to approach the true loss. Temporize with vasopressors, oxygen, head-down position, plasma expanders, etc. until the blood arrives. Remember that blood is "dynamite", each transfusion involving a calculated risk to the patient. Give enough blood to obtain a response in blood pressure or to restore 40% of the total loss within the first hour, using pressure, multiple sites, cut-downs as indicated. In a real emergency Type O Rh-negative blood can be used with due regard to the medicolegal implications.

Give oxygen for maternal shock and to improve fetal oxygenation. Prepare to resuscitate the baby. Proceed with laboratory examinations as outlined above. Proteinuria may be due to toxemia or may be a serious premonatory sign of acute renal failure.

Appraise the severity of the condition. Toxemia, an abrupt onset, a board-like continuously contracted, severely painful and tender uterus, a rapid thready pulse, respiratory distress, a falling blood pressure, a dead infant, rapid uterine enlargement, uncoagulable blood are all ominous signs.

In the second hour, do the sterile vaginal examination in the operating room under double set-up, to rule out placenta previa. Estimate how soon the patient will deliver by assessing the ripeness of the cervix, station of the presenting part, etc. Do an amniotomy immediately no matter what form of management you intend to follow and whether the fetus is alive or not. Labor usually ensues in 4 to 6 hours. Intrauterine pressure is lowered with a concommitant decrease in the amount of thromboplastin released into the maternal circulation. Moreover, the stimulation of uterine contractions helps to stop hemorrhage at the placental site.

Continue the measures instituted in the first hour. Give fibrinogen intravenously as indicated by the serial investigations outlined above. 1 liter of whole blood contains 1.2 to 1.5 gm. fibrinogen in the plasma. 1 gm. of fibrinogen elevates the circulating level by 40 mg. %. If afibrinogenemia exists 4 gm. are necessary to raise the level above 150 mg. %, therefore, give an initial dose of 2 to 4 gm. and repeat in 1 to 2 hours if required on the basis of the clot observation test. Fibrinogen is expensive and may transfer serum hepatitis.

If the fetus is still alive and vaginal delivery is anticipated within an hour (gauged by parity, ripeness and dilatation of the cervix, station and progress of the presenting part over 30 minutes), anticipate vaginal delivery which may be hastened with amniotomy and the use of low forceps. A pitocin infusion may be used for poor contractions (one 10 ml. ampoule to one liter of 5%

Dextrose in water)beginning at 10 to 20 drops per minute and gradually increasing to a minimum of 50 to 60 drops per minute. Pitocin tends to convert tetanic uterine contractions to rhythmic ones. The presence of a coagulation defect is no contraindication to the use of pitocin, which must be preceded by amniotomy. Follow the fetal heart rate carefully every 5 minutes. Should it rise over 200 per minute or become irregular, do an immediate section in the interest of the fetus. Section is also indicated if labor does not begin within 4 to 6 hours after amniotomy or if the cervix fails to dilate (no more than 5 to 6 cm. in 4 to 6 hours). Never delay longer than 10 to 12 hours. If the fetus is alive and vaginal delivery is not imminent (more than 1 hour away), do a Caesarian section, but not before shock and clotting defects are corrected.

If the fetus is dead and vaginal delivery is imminent, then proceed from below; otherwise, perform a Caesarian section for the sake of the mother when shock and coagulation defects are corrected.

Hysterectomy for *couvelaire* uterus is indicated only if the organ is so hypotonic that uncontrolable post partum hemorrhage follows. Most post-partum hemorrhages following abruption come from an apoplectic uterus.

Rupture of a Marginal Sinus

The patient is admitted to hospital and investigated as detailed above. Adequate blood replacement is given. Expectant management involves 48 hours of complete bed rest followed by 48 hours of ambulation under observation; the patient is then sent home. Definitive treatment is substituted, in the event of premature labor, recurrent hemorrhages, major hemorrhage, a rare blood type where units for transfusion are hard to obtain, a patient who cannot stay in hospital. Caesarian section is also indicated for a low-lying placenta, cephalo-pelvic disproportion or malpresentation, failure to progress in labor, fetal distress and massive hemorrhage. Otherwise vaginal delivery is performed. Labor is induced and hemorrhage staunched by amniotomy. Watch for a fore-lying cord. Pitocin may be used if indicated. Patients with recurrent hemorrhage should be managed as though this was their first hemorrhage.

THREE OBSTETRICAL EMERGENCIES

C.R. SWAYZE, 6T1.

RUPTURE OF THE UTERUS

Rupture of the uterus is a rare emergency that can occur either before or during labor. Rupture before the onset of labor is most commonly caused by rupture of a previous operative scar. (i.e. Caesarean section scar) Very rarely, rupture may occur as a result of direct trauma. Rupture during delivery can be caused by the following:

1. spontaneous rupture of an operative scar
2. spontaneous rupture of intact uterus (i.e. development of a pathologic retraction ring)
3. trauma (i.e. manipulative obstetrics)

Diagnosis: There will be sudden sharp abdominal pain, cessation of uterine contractions, rapid shock out of proportion to external bleeding, disappearance of fetal movements and heart tones and dislocation of the presenting part.

Prevention: Watch the patient with obstructed labor closely for development of a pathologic retraction ring. If a pathologic ring devlops, labor should be promptly terminated by the most conservative procedure.

Management: 1. Give immediate bloodtransfusion to correct shock.

2. Laparotomy should be performed as quickly as possible. The operation should not be delayed because of shock. In rare cases, in which there is a small tear in the uterus, repair of the wound is possible. The majority of the patients, however, require a total abdominal hysterectomy.

PRESENTATION AND PROLAPSE OF THE UMBILICAL CORD

Definition: Protrusion of a loop of umbilical cord in front of the presenting part prior to the rupture of the membranes is termed presentation; after rupture of the membranes, prolapse.

Etiology: Presentation is generally a primary condition resulting from a long cord or a low placental implantation.

Prolapse may follow a pre-existing presentation or be secondary to factors which interfere with the normal adaption of the presenting part to the lower uterine segment or to the pelvic inlet. These predisposing factors are deforemed pelvis, malpresentation (face, breech, shoulder, brow)

hydraminos, plural pregnancy, premature labor, placenta previa, premature rupture of the membranes, neoplasms altering the contour of the lower uterine segment, multiparity.

Diagnosis: Presentation of the cord is diagnosed whenever on palpation through the cervix a soft pulsating cord like body can be felt within the ammotic sac.
Prolapse is diagnosed by palpation of a loop of cord protruding through the cervix into the vagina and by signs of fetal distress in otherwise normal labor. The latter signs are:

1. irregularities of rate and rhythm of the fetal heart.
2. passage of meconium stained fluid in a vertex presentation.
3. increased activity of the fetus.

Management
Presentation: Keep the membranes intact by avoiding manual manipulation and by keeping the patient quiet. If the child is reasonably mature, cesarian section should be done.

Prolapse: If the child is dead, vaginal delivery is expected. If the child is alive, and the cervix fully dilated, immediate vaginal delivery is attempted.

If the cervix is incompletely dilated, immediate cesarian section must be done if the child is to be saved. The decision to do a section depends on the fulfilment of the following criteria:

1. The child is reasonably mature.
2. The diagnosis of prolapse was made promptly.
3. The child is alive and considered viable (strong fetal heart sounds, strong pulsation of the cord)

While preparing for the section, the patient should be placed in knee-chest position or deep Trendelenburg and a finger should be placed in the rectum or vagina to attempt to keep the presenting part out of the pelvis.

FAILED FORCEPS
Definition: An unsuccessful "all-out" attempt at vaginal delivery by forceps extraction.

Failure at forceps extraction is usually evidence of error in judgement of the part of obstetrician in dealing with one or more of the following:

1. Cephalopelvic disproportion (sufficient to contradict vaginal delivery)
2. Incomplete cervical dilatation.
3. Malposition of the fetus.

Prevention: If the patient fails to make satisfactory progress during labor, x-ray pelvimetry, and sterile vaginal examination should be done.

Forceps should be applied only if all the prerequisites for forceps application have been fulfilled.
Management:
This depends on precise evaluation of the following:

1. patient's general condition (dehydration, blood loss, infection, local trauma).
2. condition of fetus (including presentation and position).
3. cephalo-pelvic proportions.

Fluids, blood, antibiotics, and a period of rest and sedation may be required as supportive treatment.

If the cervix is not fully dilated, and there are no contra-indications to vaginal delivery, general supportive measures listed above should be instituted with anticipation of vaginal delivery when the cervix becomes fully dilated. If the infant is living, and cephalo-pelvic disproportion or abnormal presentation precluding safe vaginal delivery is present, cesarian section is indicated. If the infant is dead, destructive operations can be performed by the obstetrician, providing he has had adequate training in such procedures. Lacking such training, a cesarian section should be done.

SUICIDAL RISKS

H.A. EVANS, B.A. 6T2

Psychiatric conditions in which there is a risk of suicide include:
Depressive States
A. Reactive Depression
B. Endogenous Depression
C. Involutional Depression
Paranoid States
Hysterical Personality
Panic States

CLINICAL FEATURES
Depressive States
Reactive Depression: This condition is characterized by a pathologic dejection of mood following a traumatic experience which might naturally produce sadness. The patient complains of feeling unhappy, helpless, lost, unfit to

cope with day-to-day affairs and face the future, difficulty in concentration, lack of energy, easy fatigueability. Sleep is often disturbed and characteristically the complaint concerns difficulty in falling asleep. Very often the sleep is accompanied by unpleasant dreams. In reactive depressions the patient is usually not sad about himself; there is little self-reproachfulness but instead a tendency to accuse others — the environment and fate. External events predominate and environmental factors have a marked influence on the course of the reaction. The diagnostic feature of this type of depression is that the patient can be distracted from himself in cheerful company or at some place of entertainment and return for the time being to his normal self. The patient retains his responsiveness, can laugh at a joke and tell the physician exactly what is making him unhappy, the exact point in time when he became unhappy, and why. Somatic manifestations of depression are not a prominent feature in this state. It is the marked degree of self-pity and pre-occupation with unpleasant thoughts coupled with a sudden critical change in the environmental outlook which will precipitate a suicidal attempt.

Endogenous Depression: This condition is characterized by a depression of mood which arises "out of the blue" and is not modified by changes in the external environment. The patient complains of internal emptiness, difficulty in making decisions, loss of interest and energy. At the onset there is often an increase in *libido* but, as the depression proceeds, psychomotor retardation becomes a prominent feature. Thought content reveals delusions concerned with youthful sins, professional shortcomings and other meticulous searchings for past guilty acts. These false beliefs precipitate feelings of guilt and self-accusation.

Somatic manifestations are a prominent feature of endogenous depression. The patient appears dull and listless with a dry, sallow complexion. There is decreased sweating and failure of lacrimation and the tongue is coated. Respiration is shallow, blood pressure low, pulse rate slow, evidence of weight loss and constipation. The sleep pattern is disturbed by a premature wakening.

There is a genetic basis for this illness and the patient is described as being of the "cyclothymic personality type". Family history often reveals circular mood changes of a repetitive type, premenstrual attacks of depression, sudden appearance of compulsive activity with *joie de vivre*, variations of mood unaffected by external events and pyknic constitution.

Involutional Depression: This state of depression is associated with that period in the human life cycle when the endocrine and reproductive glands begin to suffer a decrease in function, commonly referred to as the involutional period. It is believed that with a decrease in the activity of the endocrine system there is an increased irritability of the sympathetic nervous system which triggers the symptoms. However there are psychological factors which act as threats to the individual and precipitate the condition. These are: loss of sexual function and concomitant loss of attractiveness to the opposite sex, prospect of retirement, onset of old age with accompanying insecurity because of loss of family and premature death of friends. In general the involutional period is one in which it is difficult to make new adjustments in the face of the realization that "the flower of youth has faded" together with all the great expectations of youth.

The premorbid personally reveals certain character traits which predispose the individual to this depressed state. These include compulsive symptoms, inhibition, exacting and inflexible ethical standards, lacking in humour, overconscientious, perfectionism, lack of adaptive ability, frugal or greedy, super-ego dominated, narrow in interests, chronically worrisome. The history of the premorbid personality is important in order to distinguish the involutional depression from manic depressive psychosis, late schizophrenia and senile psychosis, conditions in which there is a different prognosis.

The patient presents with a picture of profound depression, hypochondriacal complaints, extreme irritability, anxiety and agitation. The thought content reveals a narrowing of interests, delusions of sin and worthlessness, delusions of deserved imminent destruction. As the agitation increases there is weeping and wringing of the hands. The somatic manifestations include weight loss and dehydration due to anorexia or refusal to eat, cold cyanotic hands, increased pulse rate, shallow respirations, constipation and oliguria. This mental disorder has the highest risk of suicide associated with it.

Paranoid State

This condition is characterized by persistent, unalterable, systematized, logically reasoned delusions. It is a mental development which can be understood from a full knowledge of the situation and the personality of the patient and if features are present which cannot be so understood the diagnosis is in doubt.

The characteristics of the personality who becomes paranoid are: emotional immaturity and sensitivity, insecurity, stubbornness, secretiveness, obstinacy, resentfulness of discipline, lacking in humour, egotism, self-confidence, self-assertiveness, sarcasm, a drive for achievement, meticulous precision, efficiency, jealousy, un-

adaptability, inability to take criticism or suggestions, tendency to criticize readily and belittle others and "make mountains out of molehills". The patient is usually an adolescent or young adult who tells a story of some habit or circumstance associated with shame (usually homosexual tendencies) which is discovered; whereupon everyone is believed to be whispering about and avoiding him. The patient then goes to war with the world for his own self-defense. The attitude toward others is distorted by logically interconnected misinterpretations and the patient's own inclinations are mirrored in the particular motives attributed to others. Past incidents are given new interpretations and fitted into the framework of present persecution — a phenomenon known as retrospective falsification. Anger and hatred are present aggravated by ideas of reference, i.e., any stray comment is taken as a personal affront. The feeling that everyone is out to get him and that the environment is going to overwhelm him, fills the patient with anxiety which leads to a secondary depression and attempt at suicide.

Hysterical Personality

An understanding of the hysterical personality can best be grasped by a comparison with the normal child. Like the child, the hysteric is immature and egocentric. His external world is seen only in the light of how it affects him. He knows his own rights and obligations of others but forgets his duties to others. His response to emotional stimuli is brisk and appropriate but only superficial and without depth of emotion. He desires immediate satisfaction of his wishes but avoids constructive effort to gain his goal. Because of his basic narcissism, his love lacks warmth and any deep tenderness and often is sexually frigid; whereas another's love is only real when it is willing to make every sacrifice to his whims. Everything is valued in superlatives and moderation is impossible. There is no serious disturbance of deep emotions and hence trifles will initiate the stormiest situation in which the hysteric feels at home. Yet in a few minutes all the tears, accusations, insults are forgotten and the hysteric is at loss as to why others should be less able to forgive and forget. Suggestibility is a well-recognized feature of the hysterical personality and often he assumes the characteristics of speech, manner and dress of those with whom he wishes to identify. Finally, it is the desire to gain attention and win the submission of others that makes the hysteric prone to histrionic suicidal threats which he impulsively will carry out without the intention of dying.

Panic State

This is a condition of extreme fear in which the patient is overwhelmed by the stimulus, cannot discuss the situation and is either immobilized by fear or frantically overactive. The panic may result from a real external challenge but most often is caused by internal conflicts usually of a homosexual nature. The patient reacts with self-assertion, aggressiveness, overactivity or immobility. The state is usually acute and transitory but the temporary bewilderment often leads to a suicidal attempt.

ASSESSMENT

Assessment of the suicidal risk is one of the most difficult problems presented to the physician. However, a decision must be made without delay so that treatment can be initiated on an in-patient or out-patient basis.

There are certain factors considered indicative of an increased risk of suicide. These are:

1. Family history of suicide.
2. History of previous suicide attempts (including suicidal gestures).
3. Degree of self-accusation and anxiety especially in the depressive states.
4. Definite ideas on how suicide would be committed or preparatory actions eg. — purchase of a weapon or drugs, writing of farewell letters.
5. Degree of depression; in the endogenous depressions the greatest risk of suicide occurs as the patient is entering upon or coming out of depression.
6. Lack of an occupation or feeling by the patient that he is no longer of use for anything or anyone.
7. Isolation, loneliness and uprooting with the prospect of having to live with few human contacts.
8. Loss of earning power or financial worries.
9. Lack of religious ties or loss of faith in higher values.
10. Prolonged sleep disturbance.

The risk of suicide is greater in urban dwellers than in rural. Most attempts are made in the spring and the autumn. Sunday morning seems to be the most favourite time. Special caution is needed in all cases of involutional depression since serious attempts are always made and hence hospitalization is strongly urged. Since it is impossible to guard the patient completely even in hospital the best prophylaxis against suicide is a good warm doctor-patient relationship.

After the patient has discussed his suicidal impulses he should be asked to promise the doctor not to make any attempt at suicide and that he will immediately call the doctor when he feels such an impulse coming on. If the patient cannot or is unable to make such a promise then hospitalization under strict observation is indicated.

TREATMENT

Three forms of therapy are used in the treatment of the suicidal patient i.e., electroconvulsive therapy, pharmocotherapy and psychotherapy. The therapy of choice for each condition is briefly outlined:

Reactive Depression

Psychotherapy.
Tofranil.

Endogenous Depression

E.C.T.
Psychotherapy.

Involutional Depression

1. Tofranil.
2. Chlorpromazine for agitation (Largactil).
3. Sex hormones — mixed preparations containing both androgens and estrogens to relieve menopausal symptoms, such as hot flashes, sweats, tensions, uneasiness and headaches.
4. E.C.T.
5. Psychotherapy.

Paranoid State

Keep the patient under close observation and he will usually recover without treatment.

Hysterical Personality

Chlorpromazine (Largactil).
Psychotherapy.

Panic States

Barbiturates for sedation.
Psychotherapy.

ACKNOWLEDGMENT — The author expresses his appreciation to the staff of the Toronto Psychiatric Hospital for their helpful suggestions and assistance in the preparation of this paper.

A PRESCRIPTION PHARMACY DESIGNED TO MEET THE PHYSICIAN'S PROFESSIONAL NEEDS

OSBORNE'S PHARMACY

M.R. OSBORNE, Phm, B.

Ground Floor, Medical Arts Bldg.

WA. 2-2156

Our Dispensary is Open From 8:30 a.m. Until Midnight Daily

BOOK REVIEWS

Disease and the Advancement of Basic Science; edited by Henry K. Beecher. S. J. Reginald Saunders and Co.; 404 pages; $13.75.

Because this book is really a collection of the Lowell Lectures by assorted notables, it falls into the category of a "non-book" recently created by the forever-true Time magazine. The aim of this edition unfolds from the thesis that some kinds of fundamental advance in science can be made only from study of disease, for example, the understanding of the structure of the hemoglobin molecule which developed from the study of sickle cell anemia.

Twenty-one prominent scientists, most of them physicians, have contributed papers encompassing almost all fields of medical interest. Beginning with Linus Pauling's "Molecular Structure and Disease", the non-book elaborates the concept with well-done essays such as J. H. Means', "By Their Diseases Ye Shall Know Them — The Endocrines", and sublimates it at the end in Rene J. Dubos', "Medical Utopias". None of the works represent a comprehensive survey of new material. All of them are constituted of not-too-miscellaneous facts, musings, philosophies and historical tid-bits. They read easily and the reader will feel that he is listening to an expert justifying his existence, a phenomenon due, no doubt, to the origin of the papers as lectures.

This volume is not for the medical grubber or the concrete thinker but stands as very readable and enjoyable "text" for the snobbish one-upsman.

●

Textbook of Otolaryngology; De Weese, D.D., and Saunders, W.H.; The C.V. Mosby Co.; 455 pages; $8.75.

This is a first edition of a text designed primarily for the medical student and the general practitioner. Emphasis is on diagnosis and treatment. With 354 illustrations it fulfills its aim admirably. The text is succinct, easy to read and very practical. The many illustrations leave nothing in doubt. If you're not seeing the material in clinics, you're bound to find it in this book.

L. E. S.

DOWNS

SURGICAL
INSTRUMENT
MANUFACTURERS
FOR NEARLY 100 YEARS

YOUR TORONTO
HEADQUARTERS
FOR
MEDICAL SUPPLIES
OF THE
HIGHEST QUALITY

VISIT
OUR SHOWROOM
NEAR THE GENERAL HOSP.

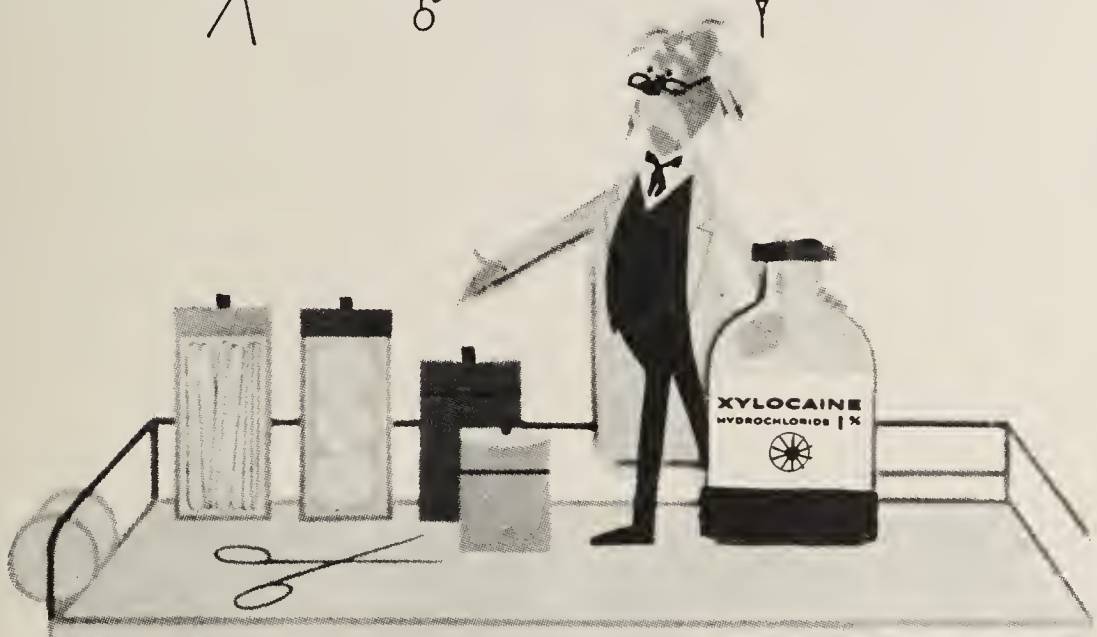
DOWN BROS. AND MAYER & PHELPS LTD.
70 Grenville Street Toronto 5, Ontario - WA. 1-8737
HEAD OFFICE: LONDON, ENGLAND

IN OFFICE SURGERY

ELECTIVE AND TRAUMATIC

use XYLOCAINE *first...*
as a local anesthetic
or a topical anesthetic

SWAB SPRAY INFILTRATION NERVE BLOCK



Xylocaine HCl solution, the versatile anesthetic for general office surgery, relieves pain promptly and effectively with adequate duration of anesthesia. It is safe and predictable. Local tissue reactions and systemic side effects are rare. Supplied in 20 cc. and 50 cc. vials; 0.5%, 1% and 2% without epinephrine and with epinephrine 1:100,000; also in 2 cc. ampules; 2% without epinephrine and with epinephrine 1:100,000.

DO YOU HAVE THE 64 PAGE MANUAL "XYLOCAINE IN ANESTHESIA" ON YOUR REFERENCE SHELF?

ASTRA *Pharmaceuticals (Canada) Ltd. Toronto 4, Ont.*

XYLOCAINE® HCl

(brand of lidocaine*)

CANADIAN PAT. NO. 503,645

MADE IN CANADA

FLAGYL

METRONIDAZOLE

potent *trichomonacide* for
ORAL and LOCAL treatment of

URETHRITIS
VAGINITIS

due to *Trichomonads*

the only anti-trichomonas conferring a
trichomonacidal power to urine and serum

oral tablets of 250 mg.
vaginal tablets of 500 mg.

Information
and samples
upon request

Poulenc LIMITED
8580 Esplanade, Montreal